MATERNAL AND FETAL OUTCOME IN JAUNDICE COMPLICATING PREGNANCY

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CHENNAI

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This is to certify that the dissertation entitled **"MATERNAL AND FETAL OUTCOME IN JAUNDICE COMPLICATING PREGNANCY"** is a bonafide work done by **Dr.M.ILANJSELVI**, in the department of Obstetrics and Gynaecology (Thanjavur Medical College) Thanjavur, in partial fulfillment of university rules and regulation for award of M.D degree in Obstetrics and Gynaecology(Branch – II) under my guidanceandsupervisionduring the academic year 2015 – 2018.

Dr. R. RAJA RAJESWARI,
M.D., DGO., Head of the Dept,
Professor,
Dept of Obstetrics and Gynaecology,
Thanjavur Medical College,
Thanjavur.

Prof. Dr. S. PRADEEPA, M.D., OG.,
Dept Obstetrics and Gynaecology,
Thanjavur Medical College,Thanjavur.

Prof. Dr. JEYAKUMAR, M.S., Mch
Dean
Thanjavur Medical College,
Thanjavur.

Thanjavur Medical College

THANJAVUR, TAMILNADU, INDIA - 613001

(Affiliated to the T.N.Dr.MGR Medical University, Chennai)

INSTITUTIONAL ETHICAL COMMITTEE

CERTIFICATE

Approval No. : 334

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MATERNAL AND FETAL OUTCOME IN JAUNDICE COMPLICATING PREGNANCY

submitted by Dr. ILANJSELVI.M **of**

Dept. of OBSTETRICS AND GYNAECOLOGY **Thanjavur Medical College, Thanjavur**

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Thanjavur

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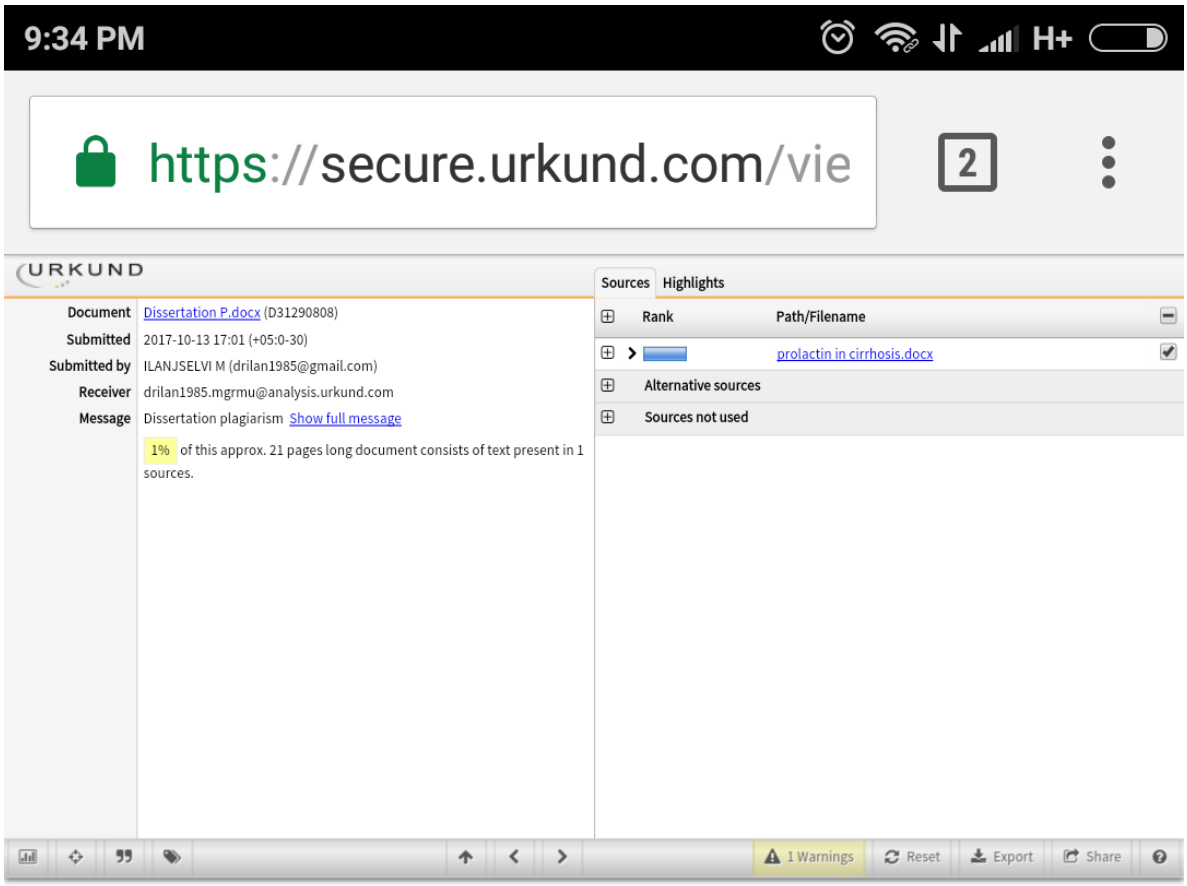

INTRODUCTION

The incidence of jaundice in India varies from 0.4 to 0.9/1000 deliveries. Jaundice in pregnancy carries a grave prognosis for both the mother and the fetus, and is responsible for 10% of maternal deaths. Liver disease in pregnancy is an important medical disorder seen more often in developing countries than in developed ones. The present study analyzes the causes and the fetomaternal outcome in pregnancies affected with jaundice. Abnormal liver test results are obtained in 3% to 5% of pregnancies because of many potential causes and the clinical outcomes ranges from self-limiting to rapidly fatal.

The main causes for abnormal liver tests in pregnant patients are: (1) Pregnancy-related liver disease. These are the common reasons for abnormal liver function tests in pregnancy. Five liver diseases unique to pregnancy includes the following - (i) Hyperemesis gravidarum (HG) (ii) Intrahepatic cholestasis of pregnancy (ICP) (iii) Preeclampsia (iv) Hemolysis, elevated liver enzymes, and low platelets (HELLP) (v) Acute fatty liver of pregnancy (AFLP). (2) Newly acquired liver diseases like acute viral hepatitis, drug induced liver injury, or gallstones (3) Preexisting chronic liver disease such as cholestatic liver disease, autoimmune hepatitis, Wilson disease, and chronic viral hepatitis. (4) physiologic changes in pregnancy - Abnormal liver function test due to physiological changes in pregnancy without liver dysfunction have a unique pattern.

The common maternal complications encountered are Encephalopathy, Disseminated intravascular coagulation , Renal failure , Shock, Postpartum hemorrhage , Pyrexia and also Death. Elevated level of serum bilirubin causes vasoconstrictive effect on the placental vessels and cardiotoxic effect resulting in fetal asphyxia and intrauterine death . Also elevated bilirubin produce cellular effect which stimulates uterine contractility and sensitizes myometrium to oxytocin resulting in preterm labour.

High maternal mortality and morbidity in our country are due to many factors like Poor hygiene, inadequate sanitation , malnutrition, prevalence of anemia, delay in seeking medical advice, lack of awareness ,and delay in referral to the higher centers. Many patients are brought in moribund condition to the hospital at admission itself and hence they do not respond to treatment.

The prevalence of viral hepatitis in pregnancy can be reduced by creating public awareness, proper sanitation facilities, safe drinking water, immunization against viral hepatitis, improved antenatal care for early detection and well equipped hospitals for intensive care . Thereby, mortality and morbidity of jaundice complicating pregnancy can be decreased.

The aim of this study is to identify the various etiologies and distribution of jaundice with reference to age, parity and trimesters and also to determine the fetomaternal outcome among the pregnant women

CERTIFICATE-II

This is to certify that the dissertation entitled "**MATERNAL AND FETAL OUTCOME IN JAUNDICE COMPLICATING PREGNANCY"** of the candidate **Dr.M.ILANJSELVI** with registration number **221516204** for the award of the degree in the branch of M.D Obstetrics and Gynaecology. I personally verified the urkund.com website for the purpose of plagiarism check. I found that the uploaded thesis file contains from introduction to conclusion pages and result shows 1 percentage of plagiarism in the dissertation.

Dr. R. RAJA RAJESWARI, M.D., DGO.,

Professor
Dept of Obstetrics and Gynaecology
Thanjavur Medical College
Thanjavur

DECLARATION

I solemnly declare that the dissertation titled **"MATERNAL AND FETAL OUTCOME IN JAUNDICECOMPLICATING PREGNANCY"** was done by me at department of Obstetrics and Gynaecology, Thanjavur Medical College during the year 2015 – 2018 under the guidance and supervision of Prof. Dr. R. RAJA RAJESWARI, M.D., DGO.,. This dissertation is submitted to the Tamil Nadu Dr. M .G. R Medical University, Chennai in partial fulfillment of the rules and regulationfor the award of M.D Degree in Obstetrics and Gynaecology (Branch – II).

Dr. M. ILANJSELVI

PLACE: Thanjavur

DATE:

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Dr. M. ILANJSELVI

CONTENTS

ABBREVIATIONS

HG: Hyperemesis gravidarum

ICP: Intr hepatic cholestasis of pregnancy

HELLP: Hemolysis,elevated liver enzymes and low platelet count

AFLP: Acute fatty liver of pregnancy

BA: Bile acid

LN: Labour natural

ABD: Assisted breech delivery

LSCS: Lower segment cesarean section

DIC: Disseminated intravascular coagulation

AKI: Acute kidney injury

ARF: Acute kidney injury

HE: Hepatic encephalopathy

WT: Weight

IUGR: Intra uterine growth retardation

MATERNAL AND FETAL OUTCOME IN JAUNDICE COMPLICATING PREGNANCY

ABSTRACT

Aim:

To evaluate the maternal and fetal outcome in patients with jaundice complicating pregnancy in a tertiary care hospital.

Methods:

The observational study was conducted in Government Thanjavur Medical College, Thanjavur, OBG department, Tamil Nadu, India. All Antenatal women with jaundice complicating pregnancy cases during one year period From August 2016 to July 2017.Different parameters including age, parity, gestational age, booking status, mode of delivery, maternal complications, Perinatal outcome in terms of birth weight, perinatal morbidity and mortality were studied.

Results:

Total cases of jaundice complicating pregnancy admitted were 65. Incidence of jaundice during pregnancy was 0.4 /1000 deliveries. Total

primigravida 61.5% and multigravida38.5%.Of this 55.9% delivered by LSCS and 45.24% delivered vaginally. Term babies were 44,preterm were 15.And 50.8%were male and 49.2% were female.Maternal complication rate24.61% was with 8 case of maternal death. Live births were 91.5% and 7.6% diagnosed as IUD

Conclusion:

Jaundice complicating pregnancy is associated with increased maternal mortality and morbidity in developing countries like India and significant role in maternal and fetal outcome.Thus it becomes necessary to create more awareness about the importance of regular antenatal care, health education, early diagnosis and appropriate timely treatment to ameliorate many cases and to bring out a satisfactory maternal and fetal outcome.

Keywords:

Jaundice complicating pregnancy, maternal outcome and fetal outcome.

INTRODUCTION

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The aim of this study is to identify the various etiologies and distribution of jaundice with reference to age, parity and trimesters and also to determine the fetomaternal outcome among the pregnant women affected by jaundice treated at GOVERNMENT RAJA MIRASDAR HOSPITAL, THANJAVUR.

HISTORY

Jaundice was once called the "morbus regius" with a belief that only the touch of a king could cure it. Jaune in French means yellow from which the word jaundice was derived.

Before 400 B.C Hippocrates has written about the yellow discoloration in association with fever. Hippocratic physicians have treated liver disease and night blindness with raw ox liver soaked in honey. In 1724 Cotton Mather has stated that “Morbus Regius, or The Royal Disease; because it brings with it the Colour of Gold unto them that have it”.

In 1864, Stadeler coined the term bilirubin. From 1862 Friedrich Theodor Frerichs is called as the father of modern liver

pathology following his publication “Klinik derLeberkrankheiten” in the year 1858.

Friedrich Theodor Frerichs

ANATOMY OF LIVER

Liver is the largest exocrine gland in the body weighing about 1.5 kg in an average adult weighing about 70kg. This is located in the right hypochondrium and a part of epigastric region.

Liver is attached to the anterior abdominal wall and the diaphragm by four distinctive ligaments:

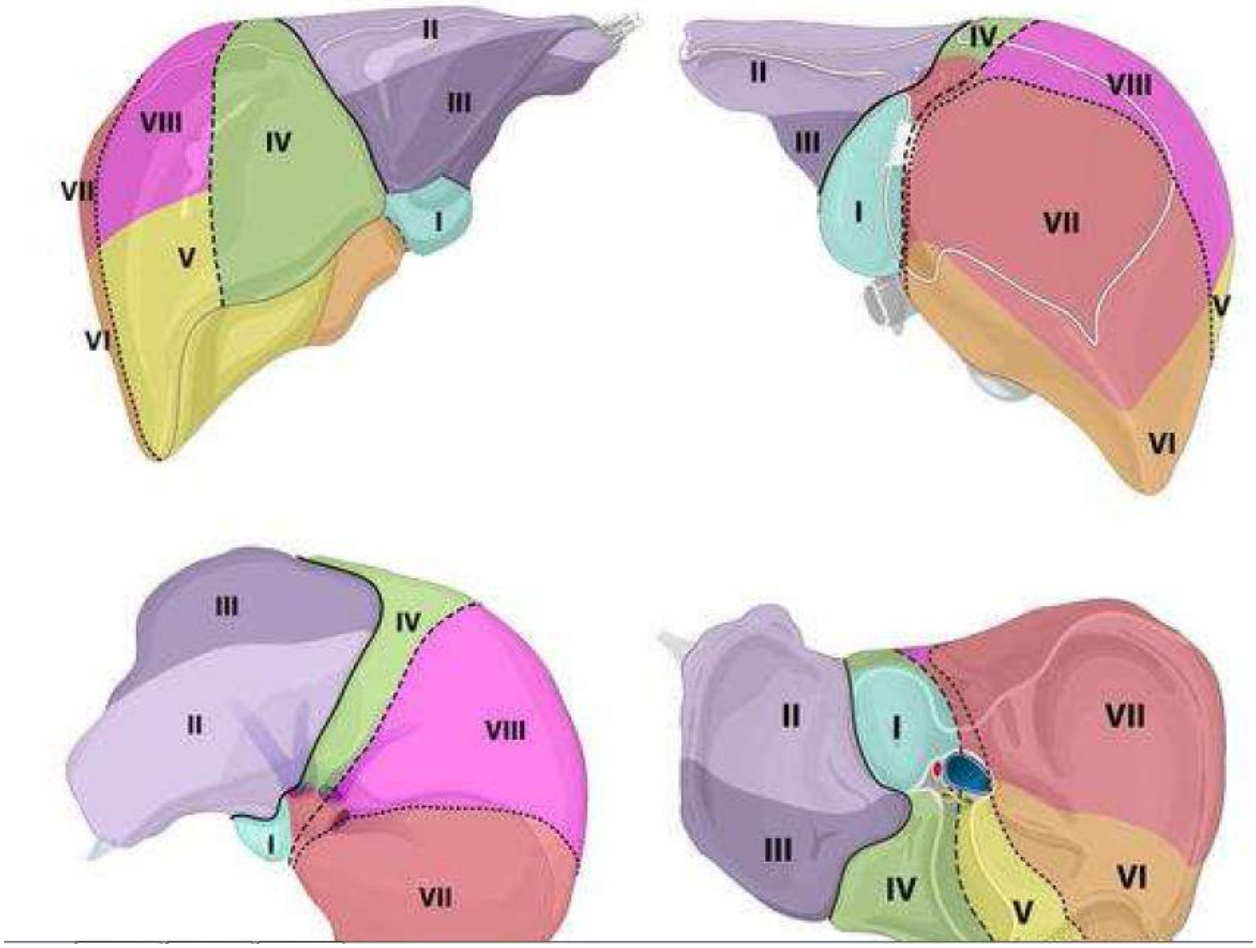
(1) Coronary ligament which connects the posterior surface of the right hepatic lobe to the diaphragm with a superior and an inferior layer between that lies in the bare area of the liver.

(2) Right triangular ligament which is formed by fusion of the superior and inferior layers of the coronary ligament.

(3) Left triangular ligament which connects the posterior surface of the left lobe of the liver to the diaphragm

(4) Falciform ligament which extends from the diaphragm and anterior wall above the level of the umbilicus to the surface of the liver, where it divides the left hepatic lobe into the left lateral and left medial segments

Liver segment is divided into eight segments. COUINAUD coined a system for liver segmental nomenclature (8 segments). Liver is divided into segments by longitudinal planes drawn through each hepatic vein to the vena cava and a transverse e level of the main portal bifurcation.Cantlie's line marks the course of the middle hepatic vein.



BLOOD SUPPLY

The two sources that supply blood to the liver are Hepatic artery and portal vein. Hepatic artery is a branch from celiac trunk of aorta . Portal vein is formed by the confluence of the superior mesenteric vein and the splenic vein at the level of the

second lumbar vertebra behind the head of pancreas. It supplies about 75% of the total liver blood supply by volume

.Blood exits the liver via the central vein accounting for 25% of cardiac output. Blood flow into the liver is controlled by number of factors like Muscular sphincters, autonomic nervous system, circulating hormones, bile salts, and metabolites.

VENOUS DRAINAGE

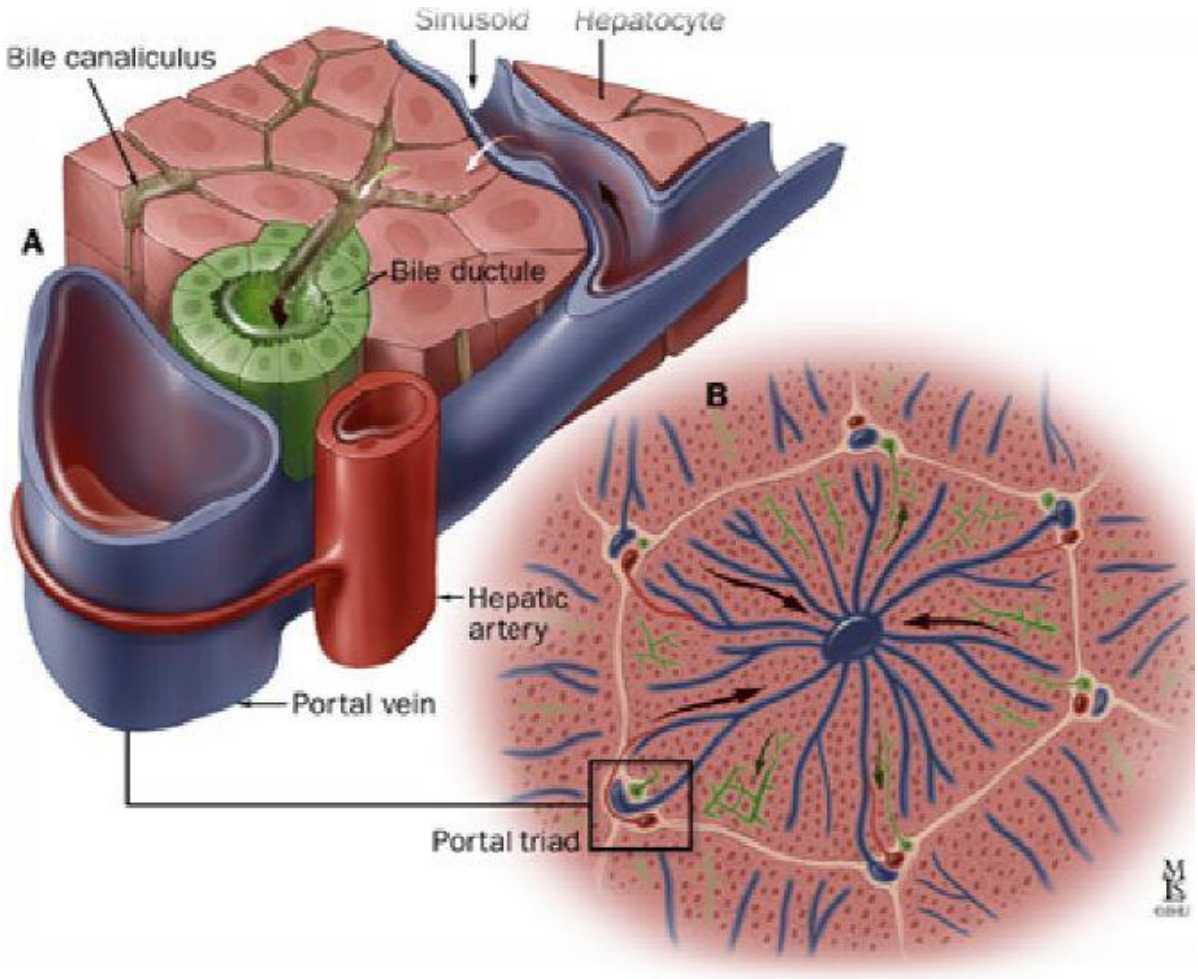
Majority of the venous drainage of the liver occurs through three hepatic veins .Right hepatic vein drains the segments 6, 7, 8 and enters directly into the vena cava. Middle hepatic vein drains segments 5 and inferior part of segment 4. Left hepatic vein drains the segments 2, 3 and superior part of 4.

MICROANATOMY

Liver is composed of hexagonal shaped units tiny hexagonal or pentagonal cylinder of about 2x1 mm. These form the anatomical units of the liver. A tributary of hepatic vein

extends through these anatomical units called interlobular vein (central vein).

Around this central vein hepatic cells are radiating outward. On the outer corners of each lobule branch of portal vein, a branch of hepatic artery and an interlobular arranged. Three adjoining drainage of bile into bile ductules of portal triad.



PORTAL TRIAD

Branches of portal vein, hepatic artery and the biliary ducts bound together in the perivascular fibrous capsule to form portal triad. Hepatocytes are arranged in plates joined with tight junctions and the apical membrane forms the biliary caniculi

Hepatocytes are segregated from the blood-filled sinusoids by fenestrated endothelial cells without a basement membrane, and by a loose connective tissue layer known as the space of Disse.

HEPATIC STELLATE CELLS

These are Star shaped cells that reside in the space of Disse. It helps to Store lipids particularly vitamin A.

Inflammatory cytokines cause activation, which involves the loss of stores of vitamin A and a dramatic upregulation in the production of extracellular matrix materials, such as collagen. When collagen is deposited in the space of Disse it impairs hepatic function.

KUPFFER CELLS

Kupffer cells belong to macrophage lineage. The sinusoids are lined by kupffer cells which clear the cellular debris and other particulate material and to some extent bacteria that enters the portal triad.

They express cell-surface receptors for altered proteins. Fc immunoglobulin receptors used to internalize foreign proteins or microorganisms that have been coated with host antibodies.

BILIARY SYSTEM

Gallbladder is a biliary reservoir that lies against the inferior surface of segments IV and V of the liver, usually making an impression against it. A peritoneal layer covers most of the gallbladder except for the portion adherent to the liver. The size is variable, but usually about 10 cm long and 3 to 5 cm wide. Gallbladder is composed of a fundus, body, infundibulum, and neck. Ultimately empties into the cystic duct

BIOCHEMICAL FUNCTIONS OF LIVER

1. Storage of substances like protein, glycogen, vitamins and folic acid.
2. Synthesis of plasma proteins, glycogen, phospholipids, bile acids and heparin.

3. Secretion of bile acids and bile pigments into the bile.
4. Metabolism of carbohydrate, protein and fat.
5. Excretion of heavy metals, hormones, cholesterol and bile pigments.
6. Detoxification of ingested drugs.
7. In fetal life the liver produces RBC and WBC.
8. Kupffer cells acts as body defence.
9. Thyroxine is converted into triiodothyronin and also participates in the activation of vitamin D.
10. Converts toxic substances into nontoxic substances e.g., benzoic acid is converted into hippuric acid by conjugation with glycine. Ammonia is converted to urea.

PRODUCTION AND METABOLISM OF BILIRUBIN:

SOURCE OF BILIRUBIN:

Mainly 80% is from senescent RBC and about 15 -20 % from ineffective erythropoiesis. Metabolism of haem containing protein can be divide into three phases:

(i) Hepatic uptake

(ii) Conjugation

(iii) Excretion into bile (rate limiting step)

UPTAKE:

Unconjugated bilirubin bound to albumin enters liver and the complex dissociates. Non-polar bilirubin enters the hepatocyte by diffusion or transport across plasma membrane. It binds to cytoplasmic anion binding protein ligandin glutathione-s-transferase and prevents efflux of bilirubin back into plasma.

CONJUGATION:

Unconjugated bilirubin is water insoluble. Hence gets conjugated to glucuronic acid forming bilirubin glucoronide , which is water-soluble.Conjugation occurs in endoplasmic reticulam. Catalysed by glucuronyl transferase in two step reaction.

EXCRETION:

Conjugated bilirubin is water soluble and is secreted by the hepatocytes into the biliary canaliculi. It is converted to

stercobilinogen by bacteria in the gut and Oxidized to stercobilin which is colored. Excreted in feces. Some stercobilin may be re-adsorbed by the gut and re-excreted by either the liver or kidney

Causes of Unconjugated hyperbilirubinaemia:

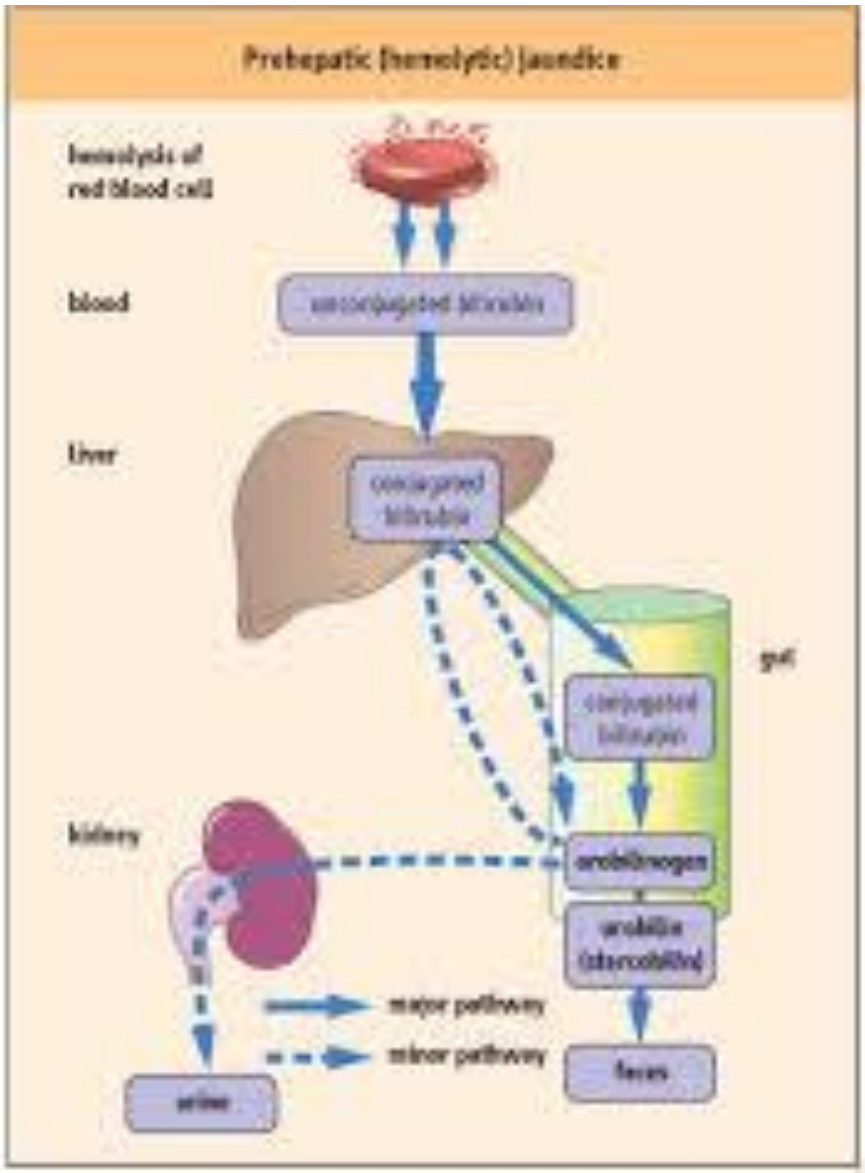
1) Increased bilirubin formation e.g., Haemolysis, Ineffective erythropoiesis, Blood transfusion and Haematoma.
2) Decreased bilirubin uptake by hepatocyte e.g., Drugs like Rifampicin, Gilbert's syndrome
3) Deficit in conjugation - Gilbert's syndrome, Crigler Najjar, Drugs.

Causes of Unconjugated hyperbilirubinaemia:

1) Dubin-Johnson syndrome, Rotor's syndrome
2) Hepatocellular dysfunction
3) Hepatic disorder with prominent cholestasis
4) Biliary duct obstruction.

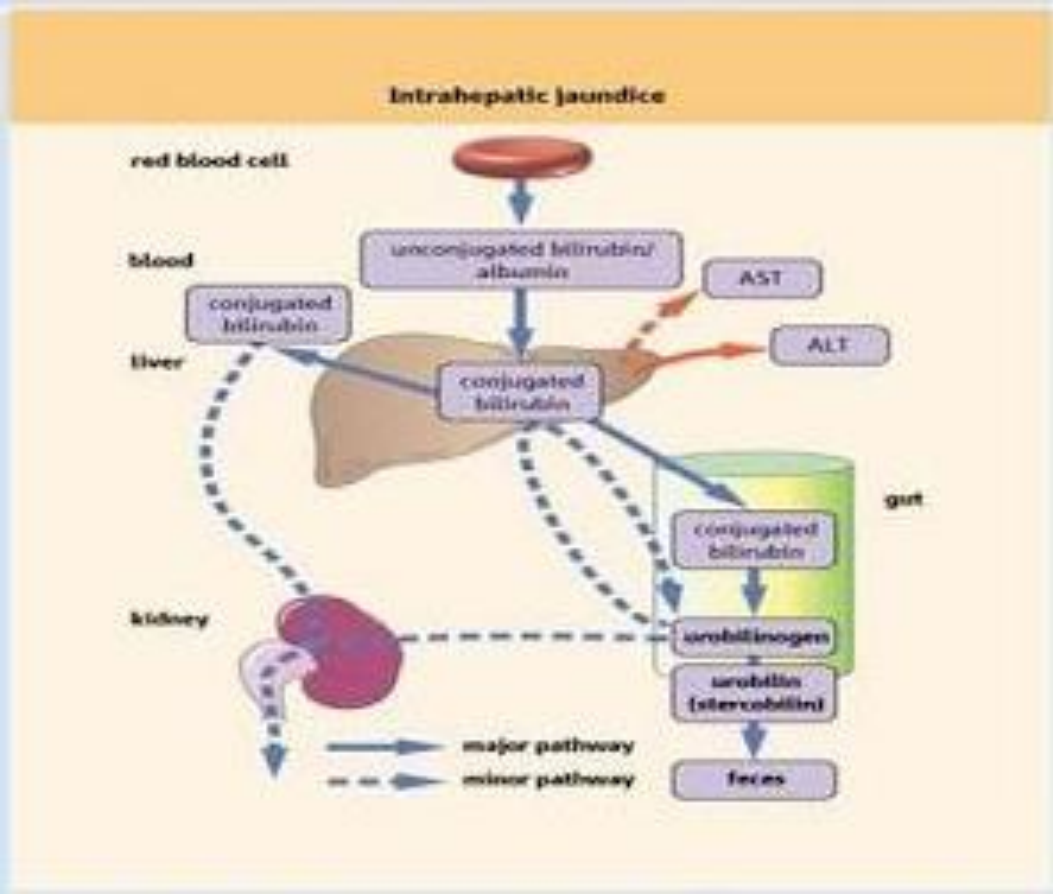
PREHEPATIC JAUNDICE

Prehepatic jaundice results from excessive RBC lysis as in haemolytic anaemia. Unconjugated bilirubin level is increased.



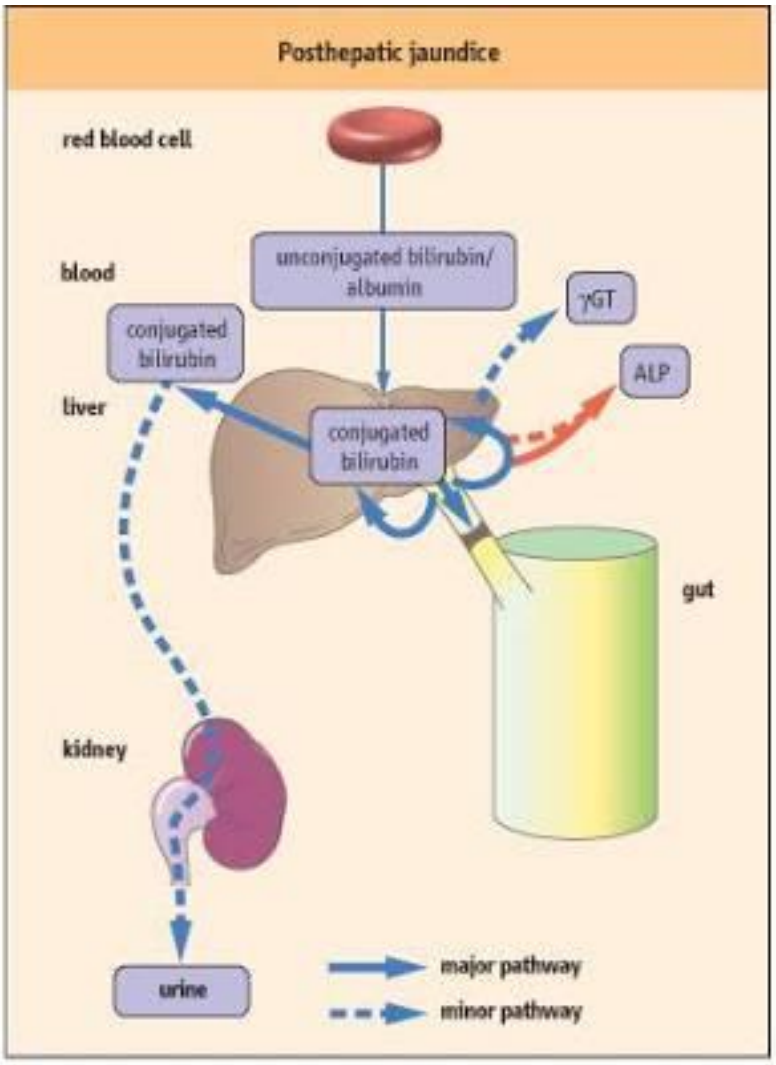
HEPATIC JAUNDICE

As a result of liver dysfunction there is impairment of uptake , conjugation and secretion of bilirubin , hence both direct and indirect bilirubin level increases.



POSTHEPATIC JAUNDICE

When there is obstruction to biliary tree, the conjugated biliribin level gets elevated.



CAUSES	JAUNDICE	BILIRUBIN IN SERUM	BILIRUBIN IN URINE	UBG IN URINE	UBG IN FAECES
Prehepatic	**Haemolytic**	**↑ indirect**	**No**	↑	↑
Hepatic	**Hepatic**	**↑Both direct and indirect**	**Yes**	↓	↓
Posthepatic	**Obstructive**	**↑Direct**	**Yes**	**No**	**No**

PHYSIOLOGIC CHANGES IN LIVER FUNCTION TESTS DURING PREGNANCY

Liver Test Results	Physiologic Changes Compared With Normal Range
Increased	Alkaline phosphatase , fibrinogen, fetoprotein, white blood cell, ceruloplasmin, cholesterol , alpha or/and beta globulins, triglycerides
Unchanged	Aminotransferases, prothrombin time
Decreased	Bilirubin, g-globulin, hemoglobin

DISEASE PRESENTATION AND THE TIMING OF ONSET DURING PREGNANCY

Disease Categories	First Trimester	Second Trimester	Third Trimester
Preexisting Liver Diseases	Chronic hepatitis B or C, autoimmune hepatitis, primary sclerosing cholangitis, Wilson disease, primary biliary cirrhosis, cirrhosis		
Newly Acquired Liver Diseases	Viral hepatitis, gallstones, drugs, sepsis, Budd-Chiari syndrome		
Diseases Related to Pregnancy	Hyperemesis gravidarum	Intrahepatic cholestasis	ICP, AFLP , preeclampsia, HELLP

AIMS
AND
OBJECTIVES

AIMS AND OBJECTIVES OF OUR STUDY:

- To analyze the maternal outcome in terms of mode of termination of pregnancy, maternal complications andmortality of jaundice complicating pregnancy.
- To identify the relation of maternal morbiditymortality in relation to admission serum bilirubin level.
- To assess fetal outcome by perinatal mortality and morbidity.
- To identify the various etiologies and distribution of jaundice with reference to age, parity and trimesters.

Inclusion criteria: Pregnant women affected by elevated liver parameters and viral positivity treated in Government Raja Mirasudar Hospital, Thanjavur.

Exclusion criteria: Normal antenatal women.

REVIEW OF LITERATURE

REVIEW OF LITERATURE

Herman Aldercreutz et al.,(1965) studied the clinical symptoms, liver function tests and ultrastructural alterations of the liver during pregnancy and found out that the ultrastructural alterations in recurrent jaundice of pregnancy seem to be rather nonspecific, since similar structural changes have been observed in biliary stasis of extra hepatic origin and in infectious or toxic liver diseases.[1]

British medical journal (1967) discussed the association between jaundice after administration of an oral contraceptive and the intrahepatic cholestasis of pregnancy. About 100 cases of jaundice from oral contraceptives have been reported.In the largest series, 50 Chilean patients, the clinical, biochemical, and microscopical findings bore a strong resemblance to those of cholestatic jaundice of pregnancy. Forty of these 50 patients had previously been pregnant, during which 17 had suffered jaundice and pruritus and ten late pruritus only.[2]

Ireneusz Roszkowski et al., (1968) studied 49 cases of jaundice in pregnancy, in which he had found out that the

presence of jaundice, regardless of origin, in pregnancy leads to a rise in acute intense contractions which shorten labor duration but increase the threat to the fetus. The course of the idiopathic jaundice in pregnancy is mild and the clinical symptoms disappear a few days after delivery. Delivery in cases of jaundice in pregnancy occurs at term and the average weight and length of the infants do not differ from those observed in normal deliveries. In idiopathic jaundice in pregnancy, complicated by pyelonephritis, premature deliveries are due to urinary tract infection rather than to the coexisting jaundice. Interrelationships between the duration of labor and the increased enzyme activity (serum transaminase, alkaline phosphatase) or increased bilirubin levels were not detected.[3]

Lunzer.,(1989) discussed about the occurrence of hepatobiliary disease with or without jaundice during pregnancy, and stated that management of liver disorders unique to pregnancy and hepatobiliary disease in general have resulted in a significant improvement in the outcome for both mother and fetus. Certain disorders such as acute fatty liver of

pregnancy and hepatic haemorrhage associated with toxaemia should be considered medical emergencies and delay in diagnosis of these conditions will probably adversely affect maternal and fetal outcome.

Thomas Murphy Goodwin., (2002) studied the strong evidence of HCG and estrogen linked with the cause of nausea and vomiting during pregnancy, and stated that the response of a pregnant woman to the primary stimulus to nausea and vomiting of pregnancy appears to depend on her susceptibility mediated by vestibular, gastrointestinal, olfactory, and behavioral pathways.[4]

Emily S. Gurley et al., (2012) estimated the population-based incidence of maternal and neonatal mortality associated with hepatitis E virus in Bangladesh, and found that 19% to 25% of all maternal deaths and 7% to 13% of all neonatal deaths in Bangladesh were associated with jaundice in pregnant women.[5]

KF Vandraas et al.,(2013) studied the association between hyperemesis gravidarum(HG) and birth outcomes in Norway which revealed no associations between HG and adverse pregnancy outcomes were observed in crude analyses, except for VPTB, Inverse associations were observed between HG and VPTB, Sub-analyses showed no associations between HG and perinatal death. It was concluded as HG was associated with slight reductions in birthweight and gestational age.[6]

AshishGoel et al., (2014) studied the Pregnancy-related liver disorders which had accounted for 8% of all maternal deaths at their center from 1999 to 2011. Of the three pregnancy-related liver disorders (acute fatty liver of pregnancy (AFLP), HELLP (Hemolysis, elevated liver enzymes, low platelets) syndrome and pre eclamptic liver dysfunction, which can lead to adverse maternal and fetal outcome, AFLP is most typically under - diagnosed. Risk of maternal death can be minimized by timely recognition and early/aggressive multi-specialty management of these conditions.[7]

Kalyani Singh., (2016) studied jaundice in pregnancy and found out that viral hepatitis was the most common cause of the jaundice and concluded that 1 in every 1000 pregnancies in India, is associated with adverse maternal and foetal outcome because of jaundice complicating pregnancy.[8]

Cihan Kaya et al., (2016) discussed about the incidence and risk factors of hyperemesis gravidarum and concluded that Non-pharmacological treatment may be offered as the first-line treatment, and Patients with electrolyte imbalance and ketonuria might require medical treatment or hospitalization.[9]

NipunVerma et al., (2017) reported that 25-year-old, 31-week pregnant woman presented with jaundice for 5 days and altered sensorium for 2 days with features of both viral acute liver failure (ALF) and tropical infections mimicking ALF, including hyperbilirubinemia, coagulopathy, anemia, thrombocytopenia, intravascular hemolysis, and hepatosplenomegaly. Etiological workup revealed rare coinfection of hepatitis E and scrub typhus which lead to poor

fetal outcome, which resulted in stillbirth and grave maternal prognosis.[10]

Anthony Laku Stephen Kirbak et al., (2017) conducted a cross-sectional study among pregnant women attending antenatal clinic for hepatitis B infection and documented that the prevalence of Hepatitis B surface antigen (HBsAg) among pregnant women attending ANC in Juba was 11%.[11]

Namrata Kumar et al., (2017) found out that Hepatitis E positivity was significantly associated with maternal mortality, intrauterine demise with prematurity, and premature rupture of membranes was the most common fetal complication, and also tabulated the distribution of different types of jaundice where Feto maternal outcomes were significantly better with HBV, HCV, and HAV hepatitis compared with HEV infection.[12]

Liver disease complicating pregnancy is divided into 3 general categories. First category includes those specifically related to the pregnancy such as acute fatty liver of pregnancy,hyperemesis gravidarum,intra hepatic

cholestasis,HELLP syndrome. Second category includes acute hepatic disorders that are coincidental to pregnancy such as acute viral hepatitis,gall stones. Third category includes chronic liver diseases. Worldwide,most common cause of jaundice is viral hepatitis.[3]

LIVER DISEASES RELATED TO PREGNANCY
HYPEREMESIS GRAVIDARUM

HG is defined as intractable nausea and vomiting during the first trimester of pregnancy. Most severe illness in the spectrum of vomiting in pregnancy.[13]In 0.3% of pregnancies it may lead to dehydration and electrolyte imbalance. But usually resolves by 16 to 18 weeks. In up to 10% of women, symptoms continues throughout pregnancy and resolve only with delivery of the fetus.[14,15]

The mechanism for Hyperemesis remains unclear. Proposed mechanisms include hormonal imbalance with elevated levels of human chorionic gonadotropin (HCG) and estrogen and decreased levels of prolactin, along with

overactivity of the hypothalamic-pituitary-adrenal axis.[16,17]High level of HCG stimulates the thyroid gland and upregulate the secretary processes of the upper gastrointestinal tract.[16] High levels of TNF alpha, IgG, IgM, C3, C4, natural killer cells, and extrathymic T cells observed in these women suggest that cytokine, T cell–mediated immune reactivation, immunoglobulin and complement play an important role in Hyperemesis.[18,19]

Risk factors includes –

a. Molar pregnancy
b. Multiple pregnancies
c. Preexisting diabetes or hyperthyroidism
d. Psychiatric disorders

Hyperemesis is a clinical diagnosis and based on exclusion of other underlying or newly acquired liver diseases.[14] An abnormal LFT is seen in up to 50% of cases.[20]Transaminases are usually 2 to10 fold elevated. Rarely can be up to 20 times the normal with mild jaundice.

HG resolves in most patients with the replacement of electrolytes and glucose, rehydration, and nutritional support. Parenteral hydrocortisone may lead to rapid resolution in severe cases, with slow improvement. Serious complications like malnutrition, esophageal tear, [21, 22] hyperthyroidism, [15]or even Wernicke encephalopathy caused by vitamin B12 deficiency can occur rarely.[23]

INTRAHEPATIC CHOLESTASIS OF PREGNANCY

Elevated bile acid (BA) levels during the late second or third trimester of pregnancy, with resolution after delivery associated with pruritus. Risk factors for ICP are advancing maternal age, twin pregnancies, multiparity and oral contraceptive use. Prevalence is 1 in 1000 to 1 in 10000.

Cause of ICP is multifactorial, involving genetic, hormonal, and exogenous factors. Sex hormones can cause cholestatic effects through inhibition of the hepatocellular bile salt export pump (Bsep).[24]Also, pregnancy is associated with an abnormal metabolic response with impaired sulfation.

The hepatic transport systems for biliary excretion are affected and saturated by the large amount of sulfated progesterone metabolites.[25,26]Genetic studies suggests that at least 10 different multidrug resistance–associated protein (MDR) 3 mutations have been identified in progressive familial intrahepatic cholestasis.[27]

Pruritus usually starts during weeks 25 to 32 of pregnancy and resolves after delivery, excoriations caused by scratching are often noted on physical examination. Serum abnormalities detected 4 weeks after the onset of pruritus.[28] Early and specific abnormality is the elevation of serum total bilirubin acid level and it is usually less than 5 mg/dL. Fetal morbidity and mortality are correlated

with maternal BA levels.

Jaundice occurs in 10%to25%of patients and some may have diarrhea. Clinical improvement in ICP with the administration of cholestyramine,[29] dexamethasone,[30] and S-adenosyl-L-methionine.

Antihistamines,benzodiazepines, phenobarbital, and epomediol have no benefit.[31]

UDCA is the treatment of choice for ICP.Close monitoring and early delivery after confirming fetal lung maturity may bethe best way to prevent sudden antenatal death. Glantz and colleagues [35]havesuggested using maternal BA levels of 40 mmol/L as a threshold for early delivery.

HELLP SYNDROME

HELLP syndrome is a multisystem disorder characterized by hemolysis, elevated levels of liver enzymes, and low platelet counts with or without preeclampsia.The prevalence is estimated to be 0.6% of deliveries.21 Risk factors are advanced maternal age, Whites and multiparity. It occurs in the third trimester in two-thirds of the Patients. Microangiopathic hemolytic anemia is associated with vascular endothelial injury, fibrin deposition in blood vessels, and platelet activation with platelet consumption. It is the hallmark of the syndrome but is not specific to this entity.

Periportal or focal parenchymal necrosis with hyaline deposition of fibrin material in the sinusoids is the characteristic histopathological finding.[33,34] Clinical symptoms includes epigastric or right upper quadrant pain, malaise, headache, nausea, and vomiting.

On physical examination, hypertension, generalized edema, and gain are common signs.

Three laboratory criteria:

- Thrombocytopenia
- Elevated aminotransferase levels
- Hemolysis. Several different classifications have been proposed.

Platelet count less than 100,000/mm3, aspartate aminotransferase levels greater than 70 U/L, and L-lactate dehydrogenase (LDH) levels greater than or equal to 600 U/L are helpful to make the diagnosis. The Mississippi classification is based on the degree of thrombocytopenia and the elevation of transaminase and LDH levels. Mississippi classification is for assessing the severity of the pathologic process. Frequent complications are disseminated intravascular coagulopathy (30%), abruptio placentae (16%), acute kidney injury (7.7%), aspiration

pneumonia (7%), pulmonary edema (6%), acute respiratory distress syndrome, cardiopulmonary arrest (4%), cerebral hemorrhage (1.2%), and retinal detachment (0.9%). Rarely, severe ascites, subcapsular hematoma, hepatic failure, and hepatic rupture can occur (0.015%).[35,36]

Proteinuria is not required to make the diagnosis. Computed tomography (CT) may show subcapsular hematomas, intraparenchymal hemorrhage, hepatic rupture, or infarction.

Transfer to a tertiary care center is advocated. If the pregnant woman is at or beyond 34 weeks' gestation or if there is any evidence of multiorgan dysfunction or severe complication, immediate induction of labor is recommended. Close monitoring of the mother should be continued after delivery also.

Most laboratory values normalize in 48 hours after delivery of the fetus. Causes of perinatal mortality include abruptio placenta,asphyxia, and prematurity. Subsequent pregnancies in patients with HELLP syndrome carry a high risk of complications including recurrent HELLP. [37, 38]

ACUTE FATTY LIVER OF PREGNANCY

AFLP occurs in 1 in 7000 to 16000 pregnancies.[39,40] Associated with microvesicular fatty infiltration of the liver, hepatic failure, andencephalopathy. It occurs commonly in the third trimester of pregnancy.Deficiencies of the enzymes of mitochondrial fatty acid beta oxidation (FAO)results in fatty infiltration of the liver. The most commonly enzyme deficiency islong-chain 2-hydroxyacyl-CoA dehydrogenase (LCHAD) deficiency. The defect isin the alpha subunit of the mitochondrial protein is associated with G1528C orE474Q mutations.[41,42]Natarajan and colleagues[43]demonstrated that placentalmitochondrial function is compromised in AFLP, which may lead to free radicalproduction and accumulation of fatty acids in the placenta, resulting in maternalhepatocyte stress and mitochondrial dysfunction leading to acute liver failure.40% to 50% of patients with AFLP are nulliparous with an increased incidence intwin pregnancies.[44]Symptoms includes anorexia, nausea, emesis, malaise,fatigue, and headache.

On physical examination, the patient may have jaundice, hypertension, edema, and hepatic encephalopathy. Serum amino transferase levels vary from 300 to 500 U/L. The total bilirubin concentration is usually less than 5 mg/dL. Other laboratory abnormalities include anemia, leukocytosis, normal or low platelet counts, coagulopathy with or without DIC, hypoalbuminemia, hypoglycemia,and acute kidney injury.

Management includes hospitalization for stabilization of hypertension and DIC, seizure prophylaxis. Fetal monitoring followed by immediate delivery of the fetus or termination of the pregnancy along with intensive support. The aminotransferase levels and encephalopathy improve within 72 hours of delivery. Most recover in 1 to 4 weeks post partum.

Maternal mortality is 3% to 12% and fetal mortality is 15% to 66%. The strong association of AFLP with LCHAD deficiency in the fetus suggests a necessity of neonatal testing for enzymatic defects of FAO. Women who are carriers of the LCHAD mutation have an increased risk of recurrence of AFLP in 20%to 70%of pregnancies.

Elevated level of serum bilirubin causes vasoconstrictive effect on the placental vessels and cardiotoxic effect resulting in fetal asphyxia and intrauterine death. Also elevated bilirubin produce cellular effect which stimulates uterine contractility and sensitizes myometrium to oxytocin resulting in preterm labour.

The fetomaternal complications of jaundice in pregnancy are not only due to jaundice alone but largely due to the underlying causes of these conditions. Thus the management depends on the underlying cause. Careful examination and vigilance is required to detect the early sign of hepatic dysfunction and to differentiate these from the physiological changes during pregnancy. Management of jaundice in pregnancy requires a combined effort of physician, gastroenterologist, obstetrician and on rare occasions, a liver transplant team.

MATERIALS AND METHODS

MATERIALS AND METHODS

Sixty five women with jaundice complicating pregnancy admitted and treated at Government Raja Mirasudar Hospital,Thanjavur from August 2016 to July 2017 were studied.

- A detailed history including patient's age, socioeconomic status , booking ,and details of menstrual history to arrive at the expected date of delivery was obtained.
- Patients were enquired in detail about their complaints and duration like nausea, vomiting , pruritus , anorexia , yellow coloured urine, pale stools, edema legs , bleeding tendency , joint pain , fever and others.
- Past history of jaundice especially in previous pregnancy and history of blood transfusion were elicited.
- systemic and obstetric examinations were carried out.
- Investigations included liver function tests, serum billirubin, SGOT, SGPT, alkaline phosphatase, Viral markers, prothrombin time (PT), partial thromboplastin

time (PTT), bleeding time (BT), clotting time (CT), platelet count and ultrasound abdomen were carried out as and when required.

- HIV screening was done in all patients.
- Medical gastroenterologist opinion was obtained for all cases.
- Labour was closely monitored. Jaundice perse was not an indication for cesarean section. Vaginal delivery with close monitoring was preferred and cesarean sections were done only for obstetric indication. After crossmatching fresh blood was kept ready as alteration in coagulation profile wasexpected in jaundice complicating pregnancy.
- Atonicity was managed with oxytocin drip, injection methergin and injection15 methyl PGF2α.
- Patients were kept in the labour ward for close observation. Clotting time wasrepeated hourly if it was prolonged till it becomes normal.
- Soon after delivery all babies were assessed by paediatrician. Alive or dead , sex , gestational age at birth ,

weight , apgar score and presence or absence of any congenital anomalies were looked for and noted. As per paediatrician opinion sick babies were admitted in preterm ward for intensive care.

- Of the 65 women, 26 had viral hepatitis, 2AFLP, 17 HELLP , 1cholestatic , 10 HELLP with severe pre eclampsia, 1 HELLP with AP eclampsia, 1 HELLP with acute kidney injury, 2 HELLP with viral infection, 3 Hyperemesis gravidarum .
- The maternal outcome was noted in terms of the mode of delivery, maternal complications and maternal mortality.The relation of maternal morbidity and mortality to the admission serum bilirubin level was analyzed
- To identify the various etiologies and distribution of jaundice with reference to age, parity and trimesters.
- Fetal outcome was assessed by perinatal morbidity and mortality

OBSERVATIONS AND RESULT

OBSERVATIONS AND RESULT

In India the incidence of jaundice varies from 0.4 to 0.9/1000 deliveries. According to this study the incidence of jaundice is 4/1000 deliveries. Kamalajayaram and Rama Devi [35]reported 0.4/1000 incidence. Singh et al [34] reported 1.03/1000 incidence while. Of the 65 women studied 56.9% were in 21 to 25 yrs of age (**TABLE 1**). Mean age is 24 yrs. About 61.5% were primi and 26.2% were Multi gravida (**TABLE 4**). 87.6% were in third third trimester (TABLE 3).

Etiologies for jaundice

Of the 65 women, 28 had viral hepatitis (43 %), 2 AFLP (3%), 17 HELLP (26.6 %), 1 cholestatic (1.5%), 3 hyperemesis(4.6%),1 haemolytic anaemia (1.5%)(**TABLE 9**). Among the 28 viral hepatitis 89.2% was due to hepatitis B, hepatitis C7.3% and hepatitis A 3.1%, 1 death due to viral hepatitice.

Relation with serum bilirubin level

Maternal mortality was directly related to the level of serum bilirubin as shown in (**Table 19**). 57 women were discharged in improved condition – 3 undelivered, 59 deliveredInitial serum bilirubin level of > 13 lead to 50% mortality. About 33.8% of women had an initial serum bilirubin level of about 5 – 10 mg / dl.

Pregnancy outcome

Among the 65 women studied 59 women got delivered (90.7%). 6 womenremained undelivered (**TABLE 5**). Of these 65 women, 4 expired due to HELLP,2 AFLP,1 AKI and 1 due to viral hepatitis. . Others improved and were discharged. Of the59 women delivered vaginal delivery (44.9%) and LSCS (55.9%).

8 cases of Atonic PPH was observed. 2 were following vaginal delivery and 6 following LSCS. .

Maternal outcome

6.1%developed hepatic encephalopathy, 28% ARF, 22% atonic PPH, 17% abruption and 5% DIC. (**Table 23**)

Cause of death

In our study, eight women expired. HELLP (50%) was the cause in 4 out of 8 women who died. Two women died of AFLP(25%) and one due to AKI (12.5%), one due to VIRAL HEPATITIS (12.5%) (**Table 26**).

Fetal outcome

0f the 59 women delivered, one was a twin delivery by labour natural survived. 91.5% born alive and 8.5 % were intrauterine death (**Table 13**). 50.8% were male babies and 49.2% were female babies (**Table 14**).

ANALYSIS

Table 1: Age distribution of the subjects in the study population

S.No	Age (in years)	Number (n)	Frequency (%)
1	≤20	7	10.77
2	21-25	37	56.93
3	26-30	15	23.08
4	31-35	5	7.69
5	>35	1	1.53

Among the studied population group (56.9%) belongs to the age group of 21-25 years,and the least common age group affected by Jaundice is >35 years of age .

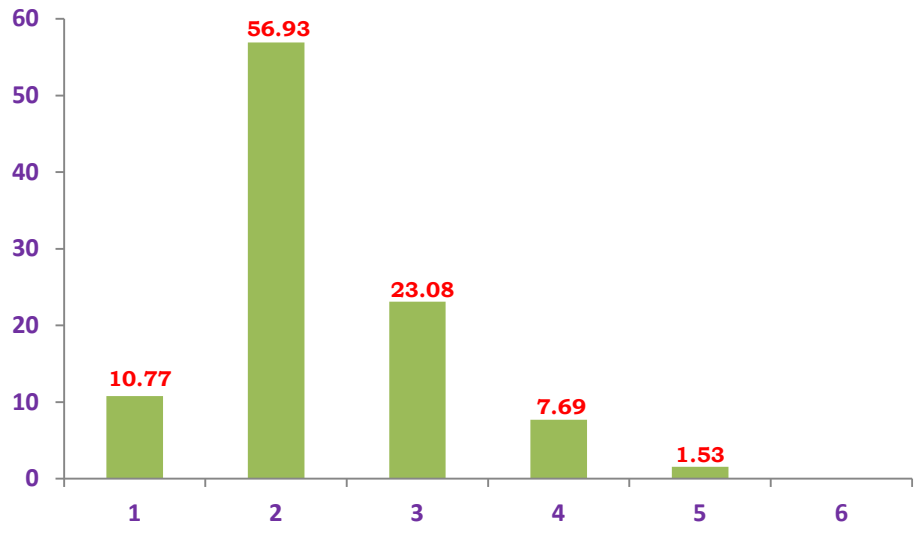


Figure.1: Age distribution of the subjects in the study population.

Table 2: Statistical data for the age analysis of the subjects in the study population

S.No	Parameter	Value
1	Mean	24.7 years
2	Median	24 years
3	Mode	24 years
4	Standard deviation	4.11
5	Variance	16.9
6	Range	19 to 43

According our study the mean age affected by jaundice is 24.7 years and thevariance is 16.9,the occurrence ranges from 19 -43.

Table 3: Trimester distribution of the subjects in the study sample

S.No	Type of the trimester		Frequency (%)
1	Trimester I	6	9.3
2	Trimester II	2	3.1
3	Trimester III	57	87.6

The occurrence of jaundice was high during third trimester. 87.6% were in thirdtrimester, lowest incidence of jaundice were seen in second trimester which was around 3.1%

	Frequency	Percent	Valid Percent	Cumulative Percent
I	6	9.3	9.3	9.3
II	2	3.1	3.1	12.4
III	57	87.6	87.6	100
Total	65	100	100	

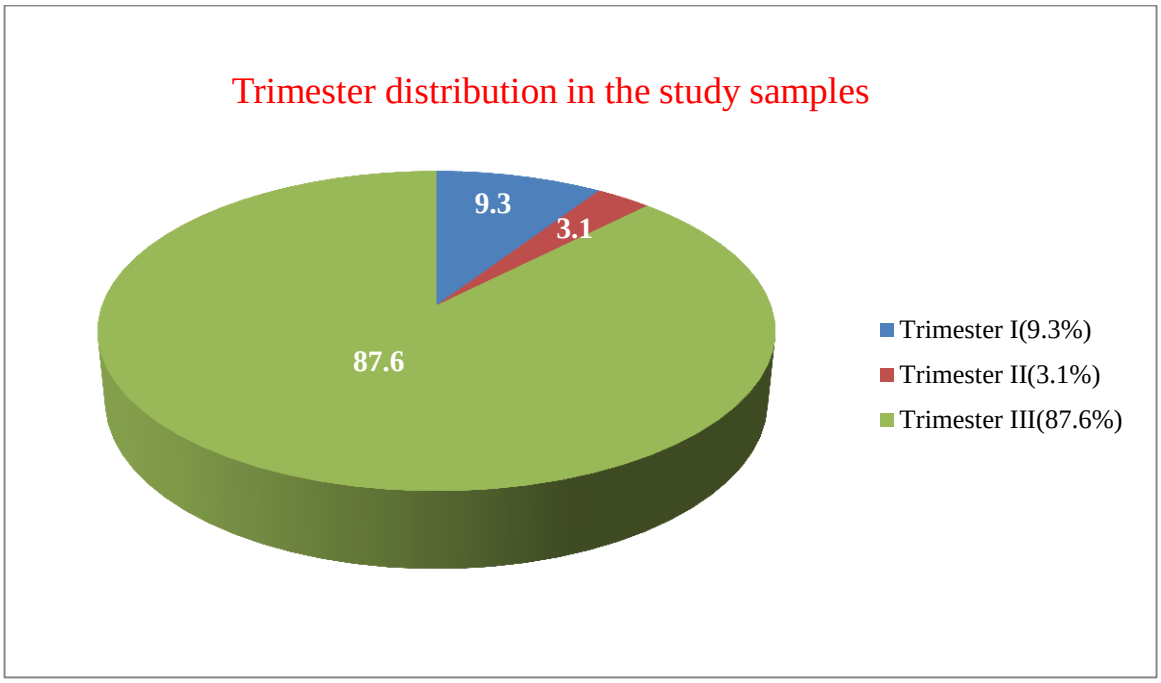


Figure 2: Trimester distribution of the subjects In the study samples

Table 4: Distribution of the type of parity in the study population

S.No	Type of parity	Number (n)	Frequency (%)
1	Primi	40	61.5
2	Gravida 2	17	26.2
3	Gravida 3	5	7.7
4	Gravida 4	1	1.5
5	Gravida 5	2	3.1

Of the total 65 women studied, 61.5% were primigravida and 26.2% were Multi gravida.

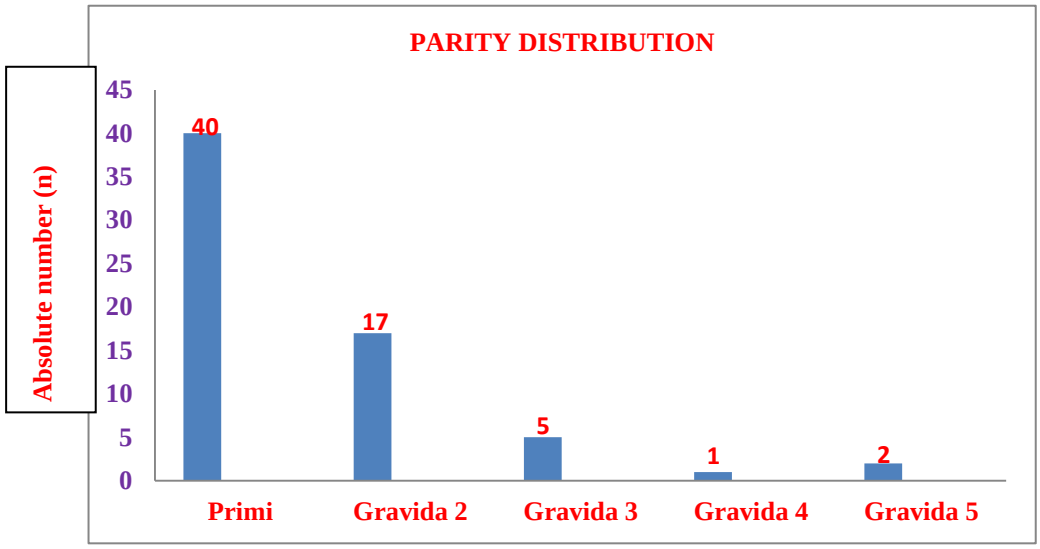


Figure 3: Distribution of the type of parity in the study population.

Of the total 65 women studied , 61.5% were primigravida and 26.2% were Multi gravida.

Table 5:Pregnancy outcome**.**

	TOTAL	**PERCENTAGE(%)**
DELIVERED	59	90.7
UNDELIVERED	6	9.3
TOTAL	65	

59 out of 65 patients delivered (90.7%). 6 remained undelivered.

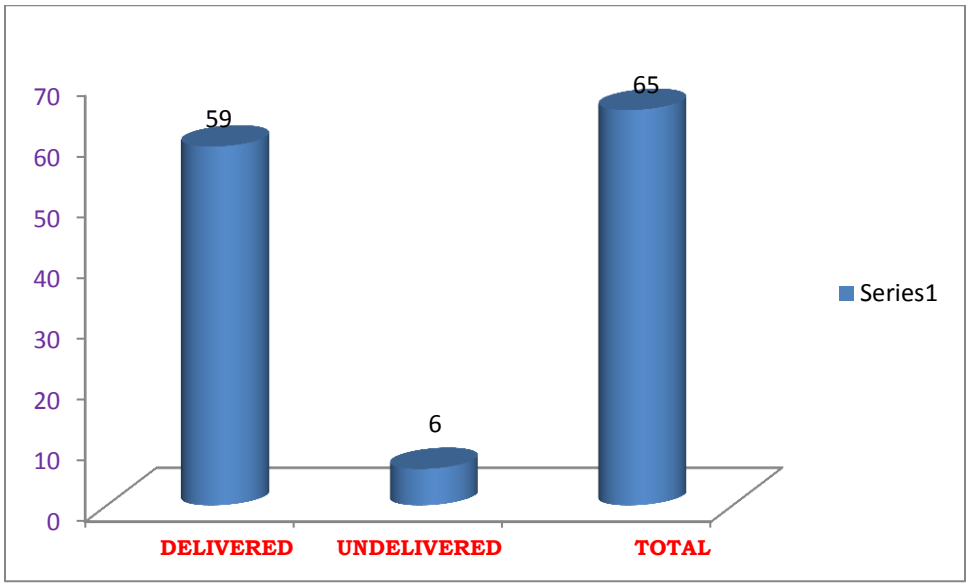


Figure 4: Pregnancy outcome

Table 6: Statistical data for the gestational age in days of the subjects in the study population

S.No	Parameter	Value
1	Mean	249.5 days
2	Median	265 days
3	Mode	270 days
4	Standard deviation	43.3 days
5	Variance	1882
6	Range	98 to 277

The mean age for occurrence of jaundice is 249.5 days and its ranges from98-277 days and the standard deviation is 43.3days.

Table 7: Distribution of the booking status and reference status of the patients in the study Population

S.No	Parameter	Number (n)	Percentage (%)
1	**Booking status**		
	Booked	63	96.9
	Not booked	2	3.1
2	**Referal**		
	Referred	60	92.3
	Self	5	7.7

From the 65 population studied, 63 patients 96.6%, were booked and only 2 patients (3.1%) were unbooked. In this study 92.3% of patients refered from various government and private hospitals and others are self.

Table 8: Frequency distribution of mode of delivery done in the study population:

S. No	Mode of delivery	Number (n)	Frequency (%)
1	ABD	1	1.6
2	Elective LSCS	2	3.3
3	LSCS	28	47.4
4	Emergency hysterotomy	1	1.6
5	Labour natural with episiotomy	23	38.9
6	RPT LSCS	2	3.6
7	Spontaneous expulsion	1	1.6
8	Vacuum	1	1.6

From the population studied, the most common mode of delivery was by LSCS around 55.9% and 44.9% were delivered vaginally.

Table 9: Frequency distribution of diagnosis done in the study population:

S.No	Type of diagnosis	Number (n)	Frequency (%)
1	AFLP	2	3.1
2	HELLP	17	26.2
3	HELLP with AKI	1	1.53
4	HELLP with eclampsia	1	1.53
5	HELLP with SPE	5	7.7
6	HELLP with viral infection	2	3.1
7	HG	2	3.1
8	HG with wernicks	1	1.53
9	Hemolytic anemia	1	1.53
10	Intrahepatic cholestatis	1	1.53
11	Partial HELLP	1	1.53
12	Viral infection	26	40
13	No definitive diagnosis attained	5	7.7

The most common case for jaundice from our study group is viral hepatitis (43.1%).

Table 10: MATURITY OF BABY

MATURITY OF BABY	TOTAL PERCENTAGE	TOTAL PERCENTAGE
PRETERM	15	24.3
TERM	44	70.9
ABORTUS	3	4.8
TOTAL	62	100

59 Babies out of15 were premature.3 out of this 59 babies were intrauterine deaths. Inspite of intensive care 2 out of 15 babies expired.

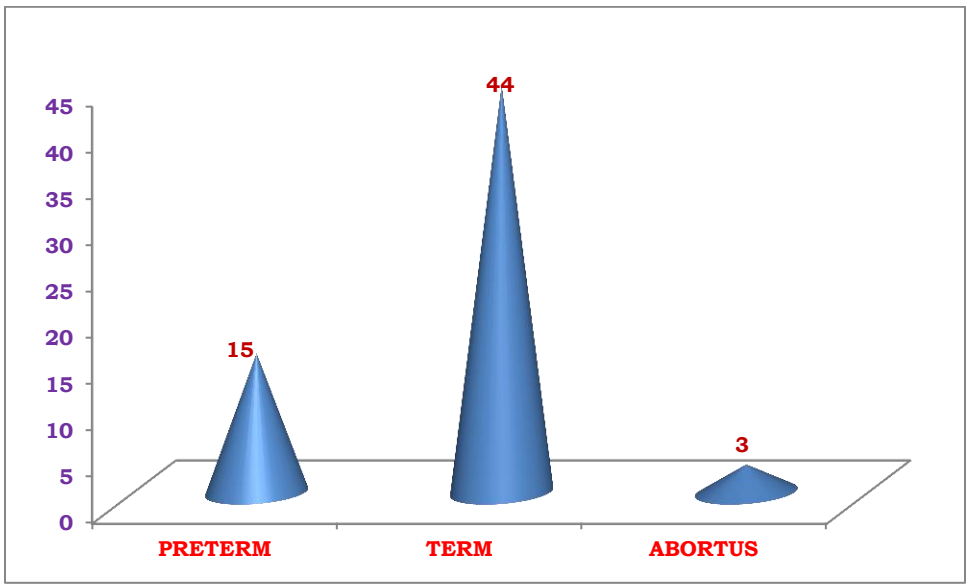


Figure 5: MATURITY OF BABY

Table 11: SEX OF THE BABY

SEX OF THE BABY	TOTAL	PERCENTAGE
MALE	30	50.8
FEMALE	29	49.2
TOTAL	59	

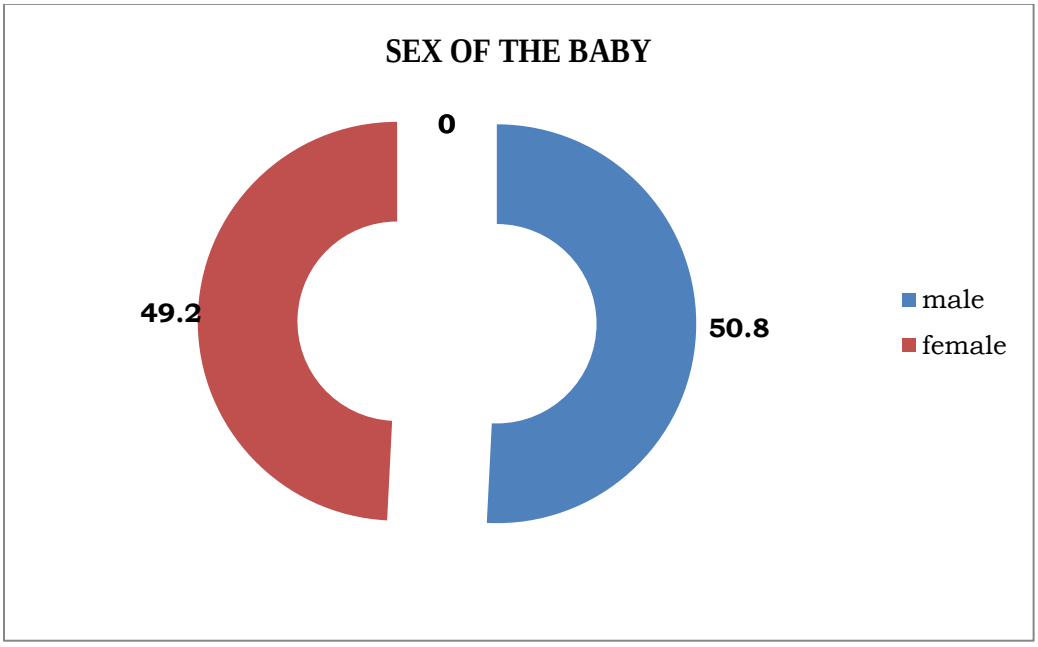


Figure 6: SEX OF THE BABY

From the population studied,(50.8%) delivered male child and 29 patients delivered female child it is 49.2%.

Table 12: Frequency distribution of neonatal outcome measures in the study population

S. No	Parameter	Number (n)	Percentage (%)
1	Term of the baby		
	Preterm	15	25.5
	Term	44	74.5
2	Birth state of the baby		
	Born alive	54	87.2
	Still born	5	8.0
	Aborted	3	4.8
3	Sex of the baby		
	Female	29	49.2
	Male	30	50.8
4	NICU admission		
	Yes	15	25.4
	No	44	84.6

Off these 59 patients 44 patients (74.5%) delivered termbabies and 15 delivered preterm babies . 87.2% were born alive and 8.5% were still born(3 IUD+ 2 PERINATAL DEATH).

Table 13: FETAL OUTCOME

FETAL OUTCOME	**TOTAL**	**PERCENTAGE**
BORN ALIVE	54	91.5
DEATH(IUD+PERINATAL)	5	8.5

0f the 59 delivered, 91.5% born alive and 8.5 % were intrauterine death.

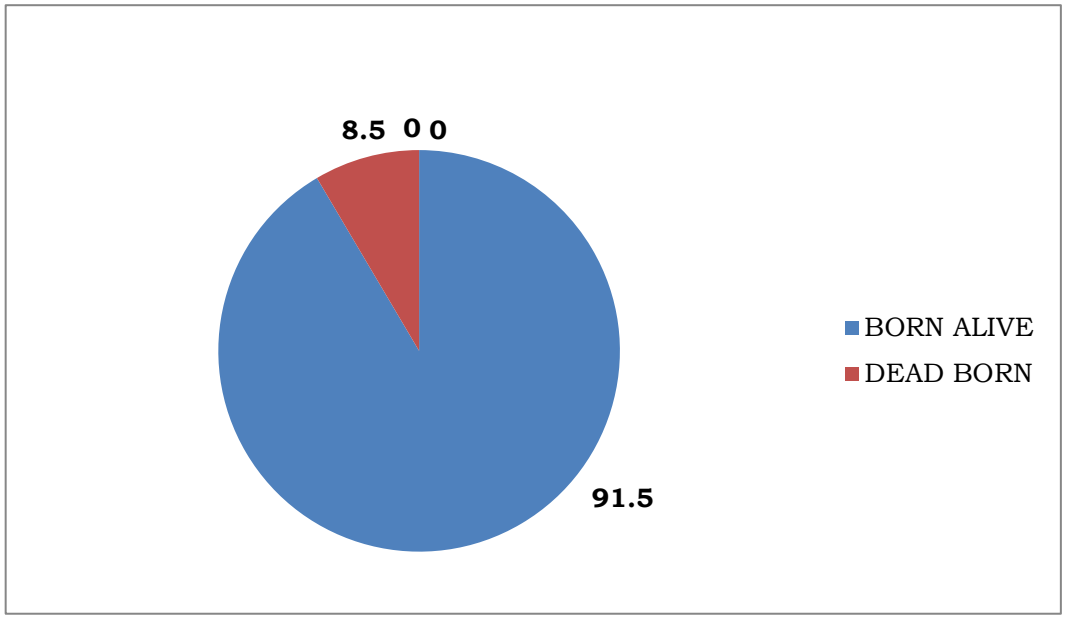


Figure 7: FETAL OUTCOME

Table 14:BIRTH WEIGHT

S.No.	Baby Weight(Kg)	Male	Female
1	<2	7	11
2	>2.5 - 3	22	18
3	>3	1	-

Total number delivered 59 of this 18 babies were low birth weight, 7 are male, and 11 were female.

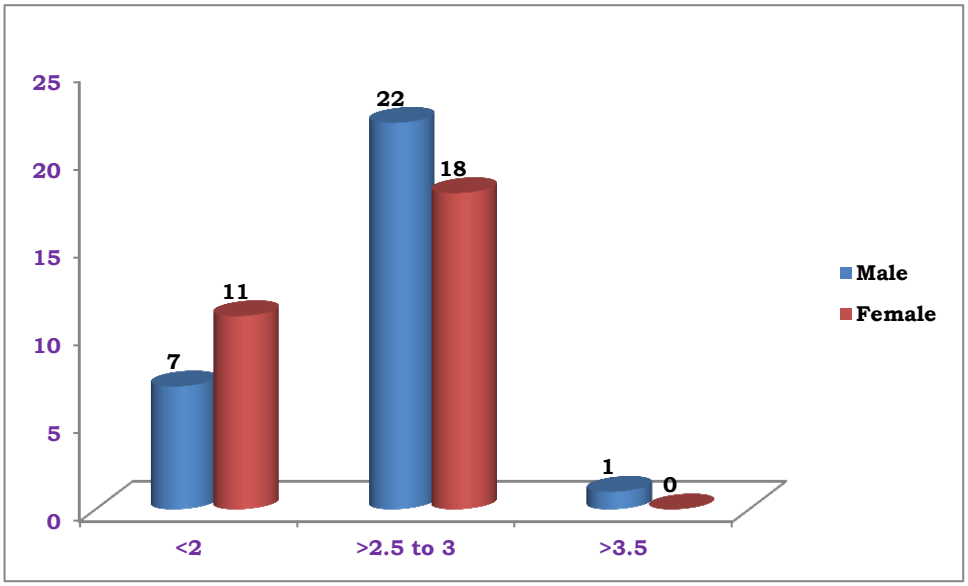


Figure 8: BIRTH WEIGHT

Table 15: Distribution of frequencies of associated medical disorders in the study population

S.No	Type of the associated medical disorders	Number (n)	Frequency (%)
1	No associated disorders	39	60
2	Acute kidney injury	1	1.5
3	Gestational diabetes mellitus	2	3.1
4	Gestational diabetes mellitus with gestational hypertension	2	3.1
5	Gestational hypertension	12	18.5
6	Gestational hypertension with severe anemia	3	4.6
7	Hypothyroidism	4	6.2
8	Nephrotic syndrome	1	1.5
9	Splenomegaly	4	1.5

The most common complication in the study group was Gestational hypertension (18.5%),least common complications are nephrotic syndrome and splenomegaly.

Table 16: Vital signs characteristics in the study population

S.No	Parameter	Mean	Standard deviation	Range
1	Heart rate (in bpm)	82.3	6.4	70 to 110
2	Systolic blood pressure (mm of Hg)	122	15.7	100 to 160
3	Diastolic blood pressure (mm of Hg)	79.2	10.6	70 to 110

In this study group the mean blood pressure of systolic 122 mm Hg and diastolic 79.2 mmHg

Table 17: Distribution of various clinical symptoms in the study population

S.No	Parameter	Number (n)	Percentage (%)
1	Icterus		
	Absent	43	66.15
	Present (+)	21	32.3
	Present (+++)	1	1.53
2	Pallor		
	Absent	34	52.3
	Present (+)	30	46.1
	Present (++)	1	1.53
3	Pedal edema		
	Absent	35	53.8
	Present (+)	20	30.7
	Present (++)	10	15.38
4	Urine output		
	Normal	47	72.3
	Decreased (↓)	11	16.92
	Decreased (↓↓)	1	1.53
	Decreased (↓↓↓)	1	1.53
	Nil	5	7.69

Of these 65 patients only 23 patients (33.8%) had Icterus others were anicteric.

Table 17a: Distribution of various biochemical parameters in the study population

S.No	Parameter	Number (n)	Percentage (%)
1	Hemoglobin (g/dl) Mean: 9.08 and SD 0.91		
	<8	4	6.15
	8-8.99	30	46.1
	9-9.99	28	43.07
	10-11	1	1.53
	>11	2	3.07
2	Platelet count		
	In normal range	20	30.7
	Decreased (↓)> 1Lack	3	4.61
	Decreased (↓↓) 50-1Lacks	1	1.53
	Decreased (↓↓↓)<50,000	41	63.07
3	Random blood sugar		
	In Normal range	62	95.38
	Decreased (↓)	2	3.07
	Increased (↑)	1	1.53
4	Renal function test		
	In normal range	61	93.84
	Elevated (↑)	3	4.61
	Elevated (↑↑)	1	1.53

The mean hemoglobin in this study group is 9-9.9 which is around 46% and 6.1% of this patients diagnosed with hemoglobin of <8gms/dl

Of these 65 patient studied 41 patient had reduced platelet count of <50,000 and 1 patient had hypoglycemia around 6% associated with elevated renal function test.

Table 17b: Distribution of various liver function test parameters in the study population

	Parameter	Number (n)	Percentage (%)
S11	Total bilirubin (mg/dl)		
	In normal range	22	33.8
	Elevated (↑)	38	58.4
	Elevated (↑↑)	4	6.15
	Elevated (↑↑↑)	1	1.53
2	InDirect bilirubin (mg/dl)		
	In normal range	21	32.3
	Elevated (↑)	40	61.5
	Elevated (↑↑)	3	4.61
	Elevated (↑↑↑)	1	1.53
3	Direct bilirubin (mg/dl)		
	In normal range	37	56.9
	Elevated (↑)	27	41.6
	Elevated (↑↑)	1	1.53
4	SGOT		
	In normal range	52	80
	Elevated (↑)	11	16.92
	Elevated (↑↑)	2	3.08
5	SGPT		
	In normal range	54	83.07
	Elevated (↑)	8	12.3
	Elevated (↑↑)	2	3.07
	Elevated (↑↑↑)	1	1.53
6	LDH		
	In normal range	64	98.46
	elevated	1	1.53
7	Bile salts and bile pigments		
	Present	2	3.07
	Absent	63	96.92
8	Bleeding time and clotting time		
	In normal range	58	89.23
	Elevated (↑)	6	9.23
	Elevated (↑↑)	1	1.53
9	Prothrombin time and INR		
	In normal range	58	89.23
	Elevated (↑↑)	7	10.76

Table 18: Distribution of viral markers in the study population

S.No	Type of viral markers	Number (n)	Frequency (%)
1	Negative	37	56.9
2	HAV positive	1	1.53
3	HBs AG positive	26	38.46
4	HCV positive	1	1.53

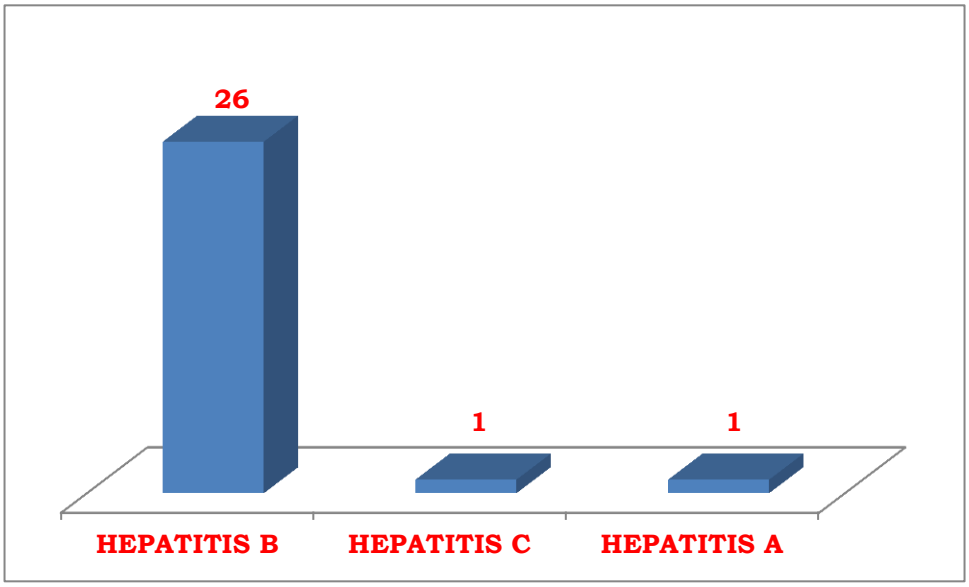


Figure 9: TYPES OF VIRAL ETIOLOGIES

Of these 28 viral Hepatitis 26 patients (86.6%) associated with Hepatitis B, 1 Hepatitis C and 1 Hepatitis A virus positivity.

Table19:LEVEL OF INITIAL BILIRUBIN

INITIAL BILIRUBIN	TOTAL	PERCENTAGE
<5	31	47.7
5-10	22	33.8
10-15	10	15.4
>15	2	3.1

Maternal mortality and morbidity was directly related to the initial level of bilirubin . Initial serum bilirubin level of > 13 lead to 50% mortality. About 33.8% of women had an initial serum bilirubin level of about 5 – 10 mg / dl.

Keeping the initial bilirubin level at admission as 10 mg/dl , the maternal outcome was poor and high mortality rate was seen when the bilirubin level exceeds 10 mg/dl. It is statistically significant.

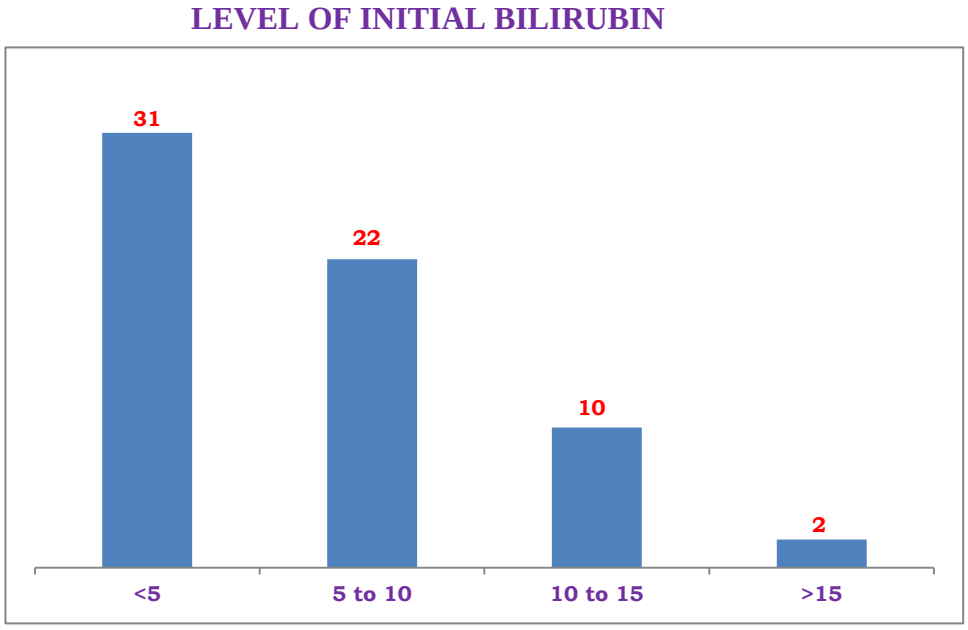


Figure 10: LEVEL OF INITIAL BILIRUBIN

Table 20: STATISTICAL SIGNIFICANCE OF INITIAL BILIRUBIN LEVEL

.	EXPIRED	RECOVERED	TOTAL
A (<10)	3	50	53
B (>10)	5	7	12
TOTAL	8	57	65
	0.123	0.008	

Initial level of bilirubin >10 mg/dl is associated with high level of maternal mortality.

Table 21: Frequency distribution of USG findings in the study population

S.No	Finding observed in USG	Number (n)	Frequency (%)
1	ABRUPTION	1	1.53
2	IUD	3	4.6
3	IUGR	10	15.3
4	Splenomagaly	1	1.53
5	Normal findings	50	77.4

During Antenatal scanning 15.3% of patients diagnosed to have IUGR babies,1 patient diagnosed as splenomegaly and 77.4% had normal antenatal scan,1patient diagnosed with abruption and 3 patients (4.1%) diagnosed with IUD.

Table 22: Maternal complications observed in the study population

S.No	Type of the complication	Number (n)	Frequency (%)
1	ABRUPTION	1	1.5
2	AKI	1	1.5
3	ATONIC	8	10.7
4	DIVC /HELLP	2	3.0
5	HEP.ENC	4	6
6	NIL	51	77.3

The most common complication encountered in jaundice during periparturn is 10.7% is PPH.

Table 23: MATERNAL COMPLICATIONS

MATERNAL COMPLICATIONS	TOTAL
HEPATIC ENCEPHALOPATHY	4
ARF	2
ATONIC PPH	8
ABRUPTION	1
DIC	2

Table 24: Description of maternal mortality outcome in the study population

S.No	**Type of outcome in maternal mortality**	**Number (n)**	**Frequency (%)**
1	Discharged	56	86.2
2	Maternal death	8	12.3
3	Discharged against medical advice	1	1.5

From our study group of 65, 56 patients (86.2%) were discharged in good condition,8 were expired, and 1paient discharged against medical advice.

Table 25: BLOOD TRANSFUSIONS

SI.No.	Number of patients	Number of packed cell	Frequency(%)
1	27	Nil	41.54
2	13	1	20.0
3	6	2	9.3
4	7	3	10.77
5	9	4	13.8
6	3	>4	4.62

According to this study 59.5% required blood transfusion,of these 20% patients transfused with 1unit only 4% of patients had >4units of packed cell transfusion.

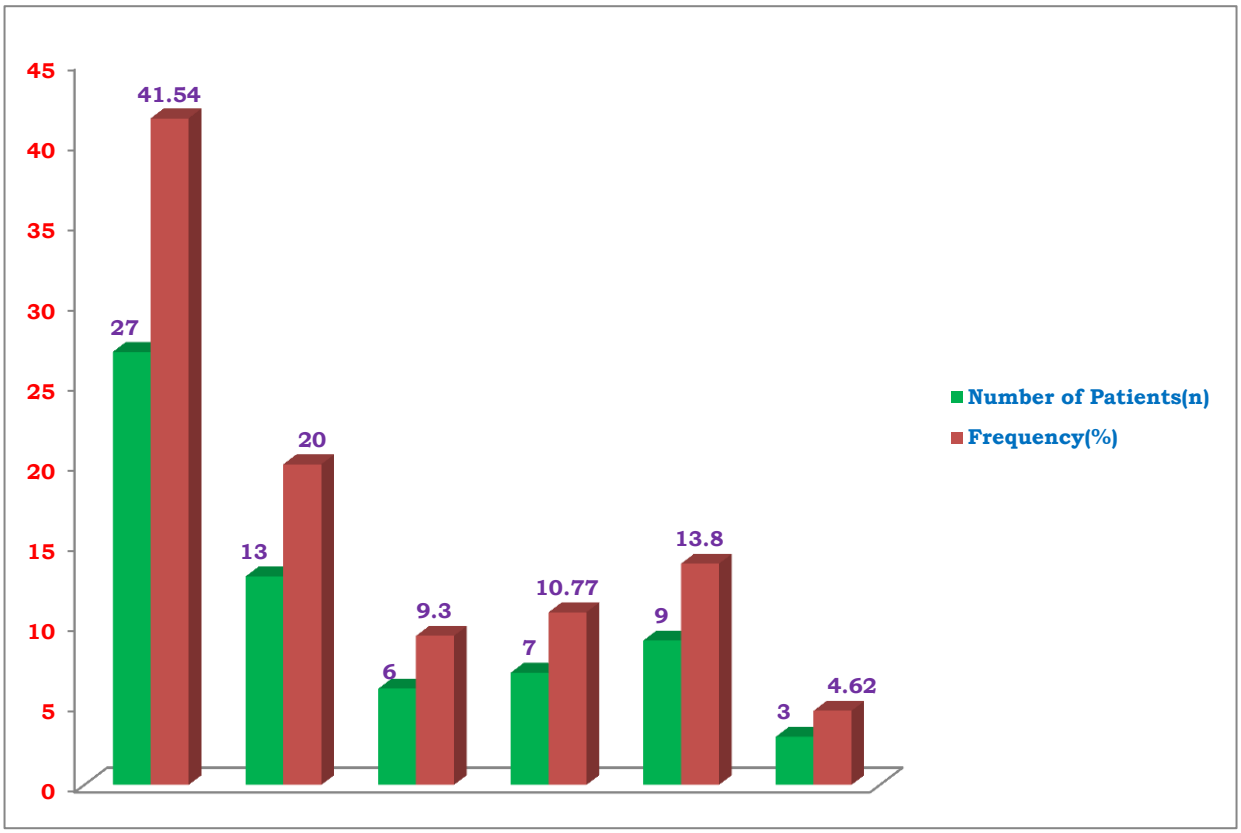


Figure 11: BLOOD TRANSFUSIONS

Table 26:CAUSE OF DEATH

CAUSE OF DEATH	Numbers	PERCENTAGE
HELLP	4	50
AFLP	2	25
VIRAL	1	12.5
AKI	1	12.5

4 women expired. HELLP (50 %) was the cause in 4 out of 8 women who died. 2 from AFLP (25%), 1 AKI (12.5%), 1 death due to VIRAL (12.5%).

CAUSE OF DEATH

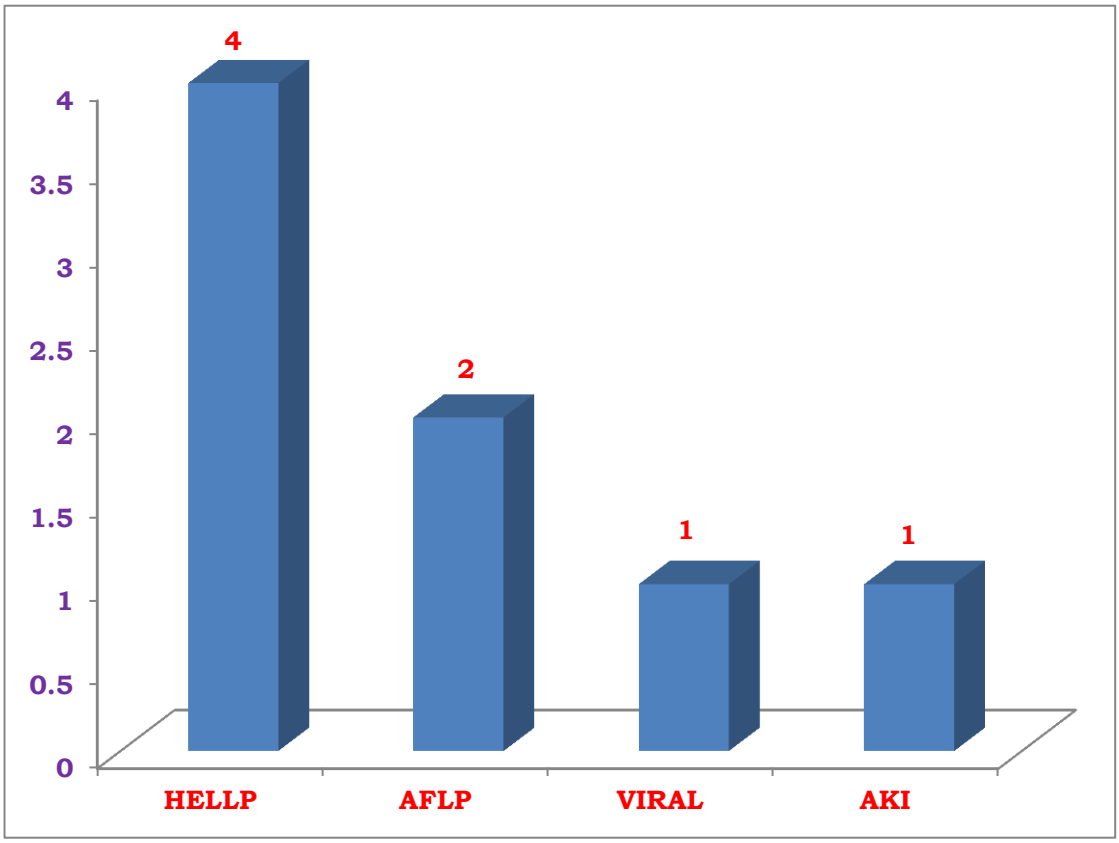


Figure 12:CAUSE OF DEATH

Table 27: COMPARISON OF NEONATAL OUTCOME.

1	Term of the baby	Preterm	15 (23.7%)	0 (0%)	p<0.0009* OR: 25.8 CI : 1.4 to 461.4
		Term	44 (37.2%)	22 (33.8%)	
2	Status of the baby	Dead	5 (7.69%)	0 (0%)	P=0.155 (NS) OR: 6.6 CI: 0.3 to 125.2
		Alive	54 (56.9%)	22 (33.8%)	
3	NICU admission	Yes	15 (6.2%)	1 (1.53%)	P=0.65(NS) OR: 2.2 CI:0.23 to21.09
		No	39(58.4%)	21 (32.3%)	

Data are expressed as absolute numbers with percentage. Fisher's exact test was used to test the statistical difference between the proportions. * indicates p value less than 0.05 and considered statistically significant.

DISCUSSION

DISCUSSION

The incidence of jaundice in India varies from 0.4 to 0.9/1000 deliveries. According to 0ur study incidence is 0.4/1OOO deliveries, which is in equal with Kamalajayaram and Rama Devi *et al*[46]reported 0.4/1000 incidence. Singh *et al*[45]reported 1.03/1000 incidence.

COMPARISON OF INCIDENCE

Previous studies	**Incidence**
Meena.N.Satia *et al*	0.81%
Brijesh J patel *et al*	0.49%
Pranithi mitta *et al*	0.31%
Nagaria Tripti *et al*	0.55%
Jayanthi *et al*	0.29%
Our study	0.4%

COMPARISON OF AGE

Previous studies	**AGE**
Meena *et al*	25-29(45.4%)
Swati *et al*	21-25(66.66%)
Brijesh *et al*	20-24(51%)
Pranithi mitta *et al*	25-29(38.09%)
Our study	21-25(56.93%)

In our study, most of the patients were in the age group of 21-25 years, which is comparable with Swati *et al*. In the study done by Meena et al,most of the patients were in the age group of 25-29 yrs. In the study done by Brijesh *et al*, most of the patients were in the age group of 20-24 years.In the study done by Pranithi mitta *et al* , most of the patients were in the age group of 21-25 years of age

COMPARISON OF PARITY

Previous studies	**Primipara**	**Multipara**
Meena *et al*	49%	47.3%
Swati *et al*	66.60%	33.40%
Pranithi mitta *et al*	38.09%	61.9%
Our study	61.50%	38.5%

In our study,61.50% were primipara.In the study done by Meena et al, 49% were primipara.In the study done by Swati *et al*, 66.6% were primipara.In the study done by Pranithi mitta *et al*,61.9% were multipara

COMPARISON OF BOOKING STATUS

Previous studies	Unbooked
Swati *et al*	93.3%
Brijesh *et al*	66%
Our study	3.1%

In our study only 3.1% were unbooked. In the study done by Swati *et al*, 93.3% were unbooked. In the study done by Brijesh *et al*, 66% were unbooked.

COMPARISON OF CAUSE OF JAUNDICE

Previous studies	HELLP	AFLP	Viral	Cholestasis	Portal HT	Hemolytic
Meena *et al*	0	0	62%	23.6%	0	0
Swati *et al*	46.3%	0	46.7%	6.7%	0%	0%
Brijesh *et al*	18.3%	0	44.8%	22.4%	0	10.2%
Krishnamoorthy *et al*	13.7%	0	50.98%	0	7.84%	0
According to this study	26.6%	3.1%	43.1%	1.53%	0	1.53%

In our study,viral hepatitis is the commonest cause of jaundice.Also in all the studies done by Meena *et al* , Swati *et al*, Brijesh *et al* and Krishnamoorthy *et al*viral hepatitis is the commonest cause of jaundice

COMPARISON OF DELIVERIES

Previous studies	**Vaginal Delivery**	**Caesarean Delivery**
Meena *et al*	81%	19%
Swati *et al*	100%	0%
Brijesh patel *et al*	82.3%	17.7%
Pranithi mitta *et al*	69.2%	30.8%
Our study	49.24%	50.76%

In our study, most of the patients were delivered by caesarean section. In all the other studies by Meena *et al*, Swati *et al* , Brijesh patel *et al* and Pranithi mitta *et al* most of the patients were delivered by labour Natural

COMPARISON OF BILIRRUBIN ON ADMISSION

Previous studies	>10(mg/dl)
Brijesh *et al*	21.4%
Pranithi mitta *et al*	14.3%
Krishnamoorthy *et al*	7.84%
Our study	15.5%

In our study, 15.5% had bilirubin >10 mg/dl at the time of admission, which is comparable with Pranithi mitta *et al study.* In the study done by Brijesh *et al,*21.4% had bilirubin >10 mg/dl at the time of admission.In the study done by Pranithi mitta et al, 14.3% had bilirubin >10 mg/dl at the time of admission. In the study done by Krishnamoorthy *et al*, 7.84% had bilirubin >10 mg/dl at the time of admission.

COMPARISON OF COMPLICATIONS

Previous studies	Multi Organ Failure	Hepatic encephalopathy	Renal failure	PPH	DIVC	Hepatic failure
Meena *et al*	9%	20%	11%	22%	44%	24%
Swati *et al*	13.3%	3.3%	13.3%	60%	20%	0
Brijesh *et al*	0	18.3%	10.2%	8.1%	26.5%	0
Pranithi mitta *et al*	0	0	7.14%	4.76 %	11.9%	0
Krishnamoorthy *et al*	0	7.87%	3.9%	9.8%	5.8	0
Our study	0	6.1%	12.5%	12%	3%	0

In our study the incidence of hepatic encephalopathy was 6.1%. In the study done by meena et al the s incidence was 20%,in Swati et al the incidence of hepatic encephalopathy was 3.3% ,in Brijesh et al the incidence of hepatic encephalopathy was 18.3%.the incidence of hepatic encephalopathy was very high in our study.on comparing with other studies the incidence of PPH is comparable with Krishnamoorthy *et al* (22%).

COMPARISON OF MATERNAL DEATHS

Previous studies	**Year**	**Percentage of deaths due to jaundice amongst total maternal deaths**
Kamalajayaram and Rama Devi	1988	12.4
Rao and Rudra	2001	15.8
Roychowdhary *et al*	1990	13.37
Bera and Sengupta	1992	19.9
Sapre and Joshi	1999	04.99
Trivedi et al	2003	29.3
According to our study	2017	12.03

In our study ,Percentage of deaths due to jaundice amongst total maternal deaths is 12.03%.it is comparable to other studies like Kamalajayaram *et al*,[46] Roychowdhary *et al*[50]and Rao *et al* [49] .

In the study done by Trivedi et al,[52] Percentage of deaths due to jaundice amongst total maternal deaths is 29.3%

COMPARISON OF CAUSES OF MATERNAL DEATHS

Previous studies	**Cause of Maternal Death**							
	HELLP	AKI	AFLP	Viral	Drug induced	Cholestasis	Pre Eclampsia	PHT
Meena *et al*	8%	0	0	59%	8%	0	0	17%
Swati *et al*	0	0	0	100%	0	0	0	0
Pranithi mitta *et al*	21.4%	0	2.38%	52.3%	0	14.2%	0	0
Krishnomoorthy *et al*	25%	0	50%	0	0	0	0	25%
Our study	50%	12.5%	25%	12.5%	0	0	0	0

In our study, viral hepatitis is the commonest cause for jaundice. It is comparable with studies done by Meena et al , Swati *et al,* Brijesh *et al* and Krishnamoorthy*et al* viral hepatitis is the commonest cause of jaundice.

HELLP syndrome is the most common cause for jaundice and associated with high mortality and morbidity.comparing with other studies the incidence of HELLP in our study 26.6%.

COMPARISON OF FETAL MATURITY

Previous studies	**Preterm**	**Term**
Meena	32-37 wks(43.6%)	31%
Swati	26.7%	73.3%
Brijesh	68.8%	31.1%
Pranithi mitta	35%	62.2%
Our study	24.3%	70.9%

In our study the fetal maturity is 70.9% which is equal with study done by Swati *et al* and Pranithi mitta *et al* majority of babies were born at or after term.In other studies by Meena *et al* and Brijesh *et al*,majority of babies were born preterm.

COMPARISON OF NEONATAL OUTCOME

Previous studies	**Live Birth**	**LBW**	**IUGR**	**IUFD**	**Preterm**	**MSAF**	**NICU Admission**
Meena et al	67%	50%	50%	22%	43.6%	28%	50%
Swathi et al	0	0	6.7%	13.5%	26.7%	40%	0
Brijesh et al	90.3%	87.7%	0	16.2%	68.8%	32.2	54.8%
Pranitha mitta et al	79.4%	85.7%	0	20.55 %	35%	0	0
Krishnamoo rthy t al	73.3%	0	0	26.6%	35%	0	0
Our study	91.5%	30%	15.3%	7.69%	23.7%	3.3%	27%

Comparing with other studies the incidence of live births(91.5%)which is comparable with Brijesh et al study which was (90.35%),incidence of low birth weight is comparable with our study is meena et al study which is (50%) ,in our study its (30%,incidence of intrauterine fetal death in our study is (7.69%),which is comparable withSwati et al study which

is(13.5%).The incidence of preterm deliveries were comparable wth Pranithi mitta study, in our study the incidence of pre term is 23.7%, and in swathi et al the incidence is26.7%. of these 27% of babies from our study were admitted NICU. which is very low with other studies.

According to our study percentage of maternal deaths due to jaundice is 12.3 % which is nearly comparable with the study conducted by Sapre and Joshi.

HELLP,AKI, AFLP and VIRAL HEPATITIS were responsible for the deaths. According to our study HELLP (50%) was the cause in 4 out of 8 women who died , two women died of AFLP,one women died of VIRAL HEPATITIS, one women died of AKI .

Various studies also report jaundice as one of the major indirect cause of maternal death, responsible for 5 to 30% of all maternal deaths [46,48-52]. Maternal deaths were directly proportional to the level of the serum bilirubin. Trivedi *et al*[52]also observed the same. According to this study the initial bilirubin level at admission > 10 is associated with poor

maternal outcome and high maternal mortality. Kamalajayaram and Rama Devi [46] reported 33.3% maternal mortality and Singh *et al*[45]reported 10%.

SUMMARY

SUMMARY

The study was conducted in 65 with jaundice complicating pregnancy. The most common age group affected by this disease are 21-25 years and its mainly found in third trimester of pregnancy.

Primi gravidas are most commonly affected by this disease. 96% of the patients were booked and immunized patients. The incidence of LSCS are more compared with vaginal deliveries, most commonest cause found in this study is viral hepatitis which is about 43.1%,1death due to hepatitis B occurred in this study.

In 59 deliveries, 15 were preterm, 3 were IUD and 2 death were during post natal period. Of this study male babies were 50.8% and female babies were 49.2% . Most common cause for jaundice in this study is HELLP syndrome, AFLP, AKI, and one death due to viral hepatitis.

The present study shows higher incidence of maternal mortality is very high in jaundice and nearly 75% patients

requires blood transfusion. DIC, Hepatic encephalopathy are the most common complications encountered in this study.

CONCLUSION

CONCLUSION

Jaundice in pregnancy is associated with high maternal mortality and perintalmortality rates.

Viral hepatitis is the leading cause of jaundice according to our study withhepatitis B being the predominant virus. Hepatic encephalopathy and acute kidney injury are the two important maternal complications. HELLP is thecommon cause of death according to our study.

According to our study the initial bilirubin level at admission > 10 isassociated with very poor maternal outcome and high maternal mortality.

The factors responsible for a high maternal mortality in our country may bedelay in seeking medical advice,poor nutrition hygiene, prevalence of anemia, and delay in referral to the hospital. Many of the patients when brought to thetertiary health care system are already in moribund condition and often, do not

respond to treatment.

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PROFORMA

PROFORMA

NAME: AGE: IPNO:

D.O.A:

D.O.DELIVERY:

D.O.DISCHARGE:

PARITY:

L.M.P: E.D.D:

COMPLAINTS:

HOPI:

PAST H/O:

MENSTRUAL H/O;

MARITAL H/O:

OBSTETRICS H/O:

ON EXAMINATION: CONSCIOUS/ORIENTED – ANAEMIC

TEMPERATURE- PEDAL EDEMA- ICTERIC – MILD/SEVERE

PR- BP- CVS- RS

P/A

P/V

INVESTIGATIONS:

HB% -

URINE ALBUMIN

SUGAR

DEPOSIT

BS/BP

BLOOD SUGAR

UREA

S.CREATININE

LFT T.BILIRUBIN

DIRECT

INDIRECT

SGOT

SGPT

ALP

LDH

PLATLET COUNT

CLOTTING TIME

BLEEDING TIME

PROTHROMBIN TIME

ACTIVATED PARTIAL THROMBOPLASTIN TIME

VIRAL MARKERS:

ULTRASOUND:

MANAGEMENT:

LIVER SUPPORTIVES

BLOOD AND BLOOD PRODUCTS TRANSFUSION:

ICU CARE:

MATERNAL OUTCOME:

MODE OF DELIVERY:

PERINATAL OUTCOME:

SEX: TERM/PRETERM: ALIVE/DEAD BORN/STILL BIRTH

APGAR 1MIN5 MIN CONGENITAL ANOMALIES:

CONSENT FORM

Study detail: A study on **"MATERNAL AND FETAL OUTCOME IN JAUNDICE COMPLICATING PREGNANCY"**

Study Centre: Department of Obstetrics and Gynaecology, Thanjavur Medical College, Thanjavur.

I confirm thatI have read and understood the information Sheet for the above study. I have had the opportunity to ask questions and all my questions and doubt have been answered to my complete satisfaction.

I understand that my participation in the study is voluntary and that I am free to withdraw at any time, without giving any reason, without my legal rights being effected.

I understand that the clinical study personnel, the Ethics Committee and the Regulatory Authorities will not need my permission to look at my health records both in respect to the current study and any further research that may be conducted in relation to in, even if I withdraw from the study. I agree to this access. However, I Understand that my identity will not be revealed in any information released to third parties or published, unless as required under the law. I agree not to restrict the use of any data or results arise from this study.

I agree to take part in the above study and to comply with the instruction given during the study and faithfully co-operate with the study team and to immediately inform the study if I suffer from any deterioration in my heath of well being or any unexpected or unusual symptoms.

I hereby give permission to undergo complete clinical examination and diagnostic tests including haematological, biochemical and radiological tests.

I hereby consent to participate in this study.
Signature/Thumb
impression:……Place……Date……
of the patient
Patient's Name, Address and Phone no: ……
Name of the Investigation: ……
Sig of the Investigator:……Place……Date……
Institution:……
Signature of the Relative/ Guardian: ……

KEEY
TO MASTER
CHART

KEEY TO MASTER CHART

GA: Gestational age

HT: Hypertension

GDM: Gestational diabetes mellitus

HB: Hemoglobin

PS: Peripheral smear

IUD: Intrauterine death

DIC: Disseminated intravascular coagulation

BS/BP: Bile salts /bile pigments

NS: Neutrophilic shift

IUGR: Intrauterine growth retardaion

MASTER CHART

	NAME	AGE	IP.NO	OBS CODE	GA(WKS)	B/UB	ASS.MED DISODER	SELF/REF	PREH/O JALINDICE	PUL RATE	BP	ICTERUS	PALLER	PE	U/O	BLOODGP	HB	PLATELET	RBS	RFT	TOT.B	I.DIRECT	DIRECT	SGOT	SGPT	LDH	
1	Radhika	25	418975	primi	37+2	B	HYPOTHYROID	REF	N	80	150/90	+	+	+	↓	A+	9.8	↓	N	N	↑	↑	↑	N	N	N	
2	Kalaivani	23	419230	primi	34+2	B	_	REF	N	88	130/90	-	+		↓	O+	8.9	↓	N	N	↑↑	↑	↑	N	N	N	
3	Vidhya	20	419320	G2P1L1	17	B	_	REF	N	78	100/60	+	+	+	↓	A+	9.5	N	N	N	↑	↑	N	N	N	N	
4	Elizhabethrani	30	433010	G2P1L1	37+3	B	GHT	REF	N	95	130/80	-	-	-	N	O+	9.8	N	N	N	↑	↑	N	N	N	N	
5	Banumathy	26	458139	primi	38+1	B	_	REF	N	82	120/80	_	_	_	N	B+	9.5	N	N	N	N	N	N	N	N	N	
6	Akila	27	458520	primi	37+4	B	GHT	REF	N	78	120/90	_	_	_	N	O+	9.8	N	N	N	N	N	N	N	N	N	
7	Tamililakkya	24	418804	primi	37	B	_	REF	N	78	120/80	+	+	+	N	O+	9	↓	N	N	↑	↑	↑	↑	↑	N	
8	Vanitha	23	434115	G2P1L1	34+1	B	GHT/ANEMIA	SELF	N	78	150/90	-	-	_	N	O+	8.9	N	N	N	↑	↑	N	N	N	N	
9	Seetha	24	433916	primi	36+1	B	GHT	REF	N	88	130/80	+	+	+	N	B+	9.9	N	N	N	↑	↑	N	N	N	N	
10	Karunambiga	21	404944	G2P1L1	18	B	_	REF	N	87	110/70	+	+	+	N	B+	11	N	N	N	↑	↑	N	N	N	N	
11	Maheshwari	25	404752	G2A1/SPE	37+5	B	SPE	REF	N	86	160/110	+	+	+	↓	O+	11.8	↓↓	N	↑↑	↑	↑	N	N	N	N	
12	Bharani	25	406216	G2A1	38+3	B	_	SELF	N	84	120/70	+	+	+	N	B+	11.8	↓↓	N	N	↑↑	↑	↑	N	N	N	
13	Dhavamani	24	456710	primi	38+2	B	_	REF	N	89	110/60	_	_	_	N	O+	8.9	N	N	N	N	N	N	N	N	N	
14	Vembu	24	459227	primi	38+4	B	_	REF	N	80	110/70	_	_	_	N	O+	8.9	N	N	N	N	N	N	N	N	N	
15	Rajeswari	27	462163	primi	38+2	B	_	REF	N	89	110/70	_	_	_	N	B+	9.1	N	N	N	N	N	N	N	N	N	
16	Maheshwari	26	462223	primi	37+5	B	_	REF	N	78	110/70	_	_	_	n	B+	8.9	N	N	N	N	N	N	N	N	N	
17	Tamililakkya	24	419804	primi	38+1	B	_	REF	N	79	120/70	+	+	+	↓	B+	9.5	N	N	N	↑↑↑	↑↑	↑	↑	↑	N	
18	Vennila	25	423218	primi	37+2	B	GHT	REF	N	82	140/90	_	_	+	N	O+	9.8	↓↓↓	N	N	↑	↑	N	N	N	N	
19	Manimegalai	24	428000	primi		B	GHT/ANEMIA	REF	N	78	120/70	+	+	_	N	B+	7.9	N	N	N	↑	↑	N	N	N	N	
20	Sharmilabanu	29	462502	G2P1L1	38+4	B	_	SELF	N	79	110/70	_	_	_	N	O+	8.9	N	N	N	↑	↑	↑	↑	N	N	
21	Kavitha	31	419320	G2P1L1	35+6	B	_	REF	N	88	120/70	_	+	+	N	A+	7.6	↓↓	N	N	↑	↑	N	N	N	N	
22	Ilakkiya	25	421576	G5P3L1	38+1	B	SPE	SELF	N	86	160/100	_	+	+	↓	B+	8.1	N	N	N	↑	↑	N	N	N	N	
23	Suganya	31	462611	G5PIL1A3	39+2	B	_	REF	N	90	110/70	_	_	_	N	O+	8.3	N	N	N	N	N	N	N	N	N	
24	Selvi	21	422687	primi	36	B	SPE	REF	N	70	160/100	_	_	++	↓	B+	8.9	N	N	N	↑	↑	↑	N	N	N	
25	Deivanai	27	428402	G2P1L1	35	B	GHT	REF	N	80	140/90	+	+	++	N	O+	8	↓	N	N	↑	↑	↑	↑	↑	N	
26	Namitha	24	422113	primi	39+2	B	_	REF	N	90	110/70	_	_	++	↓	B+	8.7	N	N	N	↑	↑	N	N	N	N	
27	Barshanibegam	21	423627	primi	39+3	B	_	REF	N	89	120/70	_	_	++	↓	O+	9.8	↓	N	N	↑↑	↑	↑	↑	↑	N	
28	Kowsalya	26	465302	G2Ai	38+4	B	_	REF	N	89	110/70	_	_	_	N	O+	9.8	N	N	N	↑	↑	↑	N	N	N	
29	Soundarya	21	466377	G2P1L1	37+5	B	_	REF	N	78	110/70	_	_	_	N	B+	9.1	↓	N	N	N	N	N	N	N	N	
30	Devika	19	471781	primi	38+4	B	_	REF	N	70	110/70	_	_	_	N	B+	9.7	N	N	N	N	N	N	N	N	N	
31	Keerthiga	29	471458	primi	38+6	B	_	REF	N	78	110/78	_	_	_	N	O+	8	N	N	N	N	N	N	N	N	N	
32	Jeya	21	471148	primi	37+5	B	_	REF	N	78	110/78	_	_	_	N	O+	8.9	N	N	N	N	N	N	N	N	N	
33	Saranya	20	472178	primi(T)	37	B	_	REF	N	76	110/70	+	+	+	N	O+	9.7			↓	N	N	↑↑↑	↑	↑	↑↑	N	
34	Banumathy	23	463548	G2P1L1	16	B	_	REF	N	80	110/70	_	+	_	N	B+	8	N	N	N	N	N	N	N	N	N	
35	Vembu	20	475529	Primi	38+2	B	-	REF	N	80	110/70	_	_	_	N	O+	9.5	N	N	N	N	N	N	N	N	N	
36	Dhanalaksmi	21	450693	primi	37+1	B	AKI	SELF	N	89	110/80	_	_	_	↓	B+	9.6	N	↑	↑	↑	↑	N	N	N	N	
37	Kala	28	480831	primi/lud	34	B	GDM	REF	YES	78	150/90	+	+	+	+	O+	9.7	↓	↓	N	↑↑	↑↑	N	↑	↑	N	
38	Dhanalaksmi	29	433648	G2P1L1	39	B	GDM/GHT	REF	N	110	110/78	_	_	_	_	AB+	8.9	N	N	N	↑	↑	N	N	N	N	
39	Jeevitha	32	431887	G2P1L1	38+4	B	GDM	REF	N	89	120/70	_	_	_	_	O+	9.2	N	N	N	↑	↑	N	N	N	N	
40	Jenifer	19	462254	G3P2L1	16	B		REF	N	78	120/80	+	+	+	↓↓	O-VE	8.1	↓	N	↑	↑	↑	↑	↑	↑	N	
41	Jeevitha	20	477848	primi	38+2	B		REF	N	87	160/110	_	_	_	_	A+	9.8	N	N	N	N	N	N	N	N	N	
42	Ilakkiya	22	435342	primi	37+6	B	SPE	REF	N	87	156/90	_	_	++	N	O+	7.9	N	N	N	↑	↑	N	N	N	N	
43	Anjanimal	24	479848	G2P1L1	38+5	B	GHT	REF	N	87	110/80	_	_	_	N	B+	8.8	N	N	N	N	N	N	N	N	N	
44	veerammal	22	481321	primi	38+6	B	_	REF	N	88	120/80	+	+	++	N	O+	8.9	↓	N	N	↑	↑	↑	N	N	N	
45	Radhika	25	480189	primi	39+1	B	_	REF	N	87	110/78	_	_	_	_	O+	8.6	N	N	N	N	N	N	N	N	N	
46	Vetriselvi	26	483117	G3P1L1P	38+2	B	_	REF	N	78	110/80	+	+	++	N	O+	9.8	↓	N	N	↑	↑	↑	N	N	N	
47	Deepa	26	467836		39+2	B	GHT	REF	N	82	130/90	+	+	+	N	B+	9.2	↓	N	N	↑	↑	↑	N	N	NS+TCP	
48	Gowtharni	25	481948	primi	39+1	B	neph.syndrome	REF	N	80	110/80	_	_	+	+	AB-VE	9	↓	N	N	↑	↑	↑	↑↑↑	↑↑↑	N	
49	Suganya	19	479219	primi	38+4	B	GHT	REF	YES	78	130/90	+	+	+	↓	O+	8.9	↓	N	N	↑	↑	↑	↑	N	N	
50	Nathiya	26	490312	G3P1L1	27	UB	GHT	REF	N	87	140/90	+	+	_	N	A+	9	↓	N	N	↑	↑	↑	N	N	N	
51	Shanthy	23	458080	primi	14	B	_	REF	N	80	110/80	+	+	_	N	o_ve	8	N	N	N	↑	↑	↑	N	N	N	
52	Vembarasi	27	477081	primi	37+1	B	_	REF	N	70	110/80	_	_	_	N	o_ve	8.8	N	N	N	N	N	N	N	N	N	
53	Manjula	25	478579	primi	38+6	B	_	REF	N	77	110/70	_	_	_	N	A+	8.8	N	N	N	N	N	N	N	N	N	
54	Fathima begam	43	478659	G3P2L1	32	UB	GHT	REF	YES	78	130/90	+	+	_	↓↓↓	B+	8.1	↓	N	N	↑	↑	↑	N	N	N	
55	Sarasu	23	48919	G2P1Li	37+6	B	_	REF	N	78	110/70	_	_	_	N	O+	8.7	N	N	N	N	N	N	N	N	N	
56	Devi	32	48392	G4P3L3	25	B	_	REF	N	76	110/80	+	+	+	N	A+	8.2	N	N	N	↑	↑	↑	N	N	N	
57	Soundarambal	21	483948	primi	36+4	B	GHT	REF	N	78	130/80	_	_	_	N	O+	8.9	↓	N	N	↑	↑	↑	N	↑	N	
58	Pavithra	24	483779	primi	34+2	B	_	REF	N	80	120/70	_	+	+	N	o_ve	9	↓	N	N	↑	↑	↑↑	↑↑	↑↑	N	
59	Boopathy	34	484358	G2PIL1	38+4	B	_	REF	N	80	120/80	+++	+	_	N	O+	8.9	N	N	N	↑	↑	↑	↑	↑	N	
60	Selvarani	21	484456	primi	38	B	_	REF	YES	80	120/90	_	+	_	N	O+	8.9	N	N	N	↑	↑	N	N	N	N	
61	Tamilarasi	21	485028	primi	37+3	B	_	REF	N	79	110/70	_	_	_	N	O+	8.9	N	N	N	N	N	N	N	N	N	
62	Nagalakshmi	22	490561	primi	37+6	B	GHT/ANEMIA	REF	N	79	120/70	_	++	++	N	O+	7.9	↓	N	N	↑	↑	↑	N	N	N	
63	Lakshmi	25	491651	primi	39+4	B	GHT/GDM	REF	N	80	140/90	_	+	++	N	B+	9.7	↓	N	N	↑	↑	↑	N	N	N	
64	Banu	24	491678	primi	38	B	GHT	REF	N	89	130/80	_	+	++	N	O+	9.8	N	N	N	↑	↑	N	N	N	N	
65	Nandhini	25	491417	G3P2L1	39	B	_	REF	N	88	110/80	_	_	_	_	O+	9	N	N	N	N	N	N	N	N	N	

BS/BP	BT/CT	PT-INR	VIRAL MARKERS	EVIDENCE OF HEMILYSIS ON PS	USG	URINES/A	NAME	DIAGNOSIS	IODE OF DELIVERY	INDICATION	BLOOD	TAL OUTCOME T/PT	AGE/SEX	WEIGHT	NICU,AD	COMPLICATION	REF.TO	M.DEATH	
+	↑↑	↑↑	NEG	YES	NORMAL	NIL	Radhika	HELLP	LSCS	FD/DIVC	3PC+4FFP	T	A/MCH	2.5		HEP.ENCEP	NO	DEATH	HELLP
NEG			NEG	NO	NORMAL	NIL	Kalaivani	HELLP	LSCS	FD/DIVC	3PC+10FFP	PRETERM	A/FCH	2.1		DIVC	NO	NO	
NEG			NEG	NO	NORMAL	NIL	Vidhya	HG			1PC		-		-	-	-	NO	
NEG			NEG	NO	NORMAL	NIL	Elizhabethı	HELLP	LSCS	PLSCS	-	T	A/MCH	2.1	-	-	-	-	
NEG			NEG	NO	NORMAL	NIL	Banumathı	HELLP	LN/EPI		-	T	A/MCH	3.1		_	NO	NO	
NEG			HCV+VE	NO	NORMAL	NIL	Akila	VIRAL	LN/EPI		_	T	A/FCH	2.8		_	NO	NO	
NEG	↑↑	↑↑	NEG	YES	NORMAL	NIL	Tamillakky	AFLP	LN/EPI		5PC+10P+25FFP	T	A/FCH	2.7	_	HEP.ENCEP	NO	YES	
NEG			HBsAg+VE	NO	NORMAL	NIL	Vanitha	VIRAL	LSCS	PLSCS		PT	A/MCH	1.5	YES	_	_	NO	NO
NEG			HBsAg+VE	NO	NORMAL	/+++	Seetha	VIRAL	LSCS	UF CX	3PC+2FFP	PT	A/MCH	2.1		ATONIC		NO	NO
NEG			NEG	NO	NORMAL	NIL	Karunambi	HG/WERNICS	_	_	_	_	_	___	_	_	_	YES	NO
NEG			NEG	NO	NORMAL	/+++	Maheshwa	_	LSCS	FD	1PC+4FFP	T	A/MCH	2.5	_	_	_	YES	NO
NEG			NEG	NO	NORMAL	/+++	Bharani	HELLP	VACCUM		4PC+12FFP	T	A/FCH	2.5				YES	NO
NEG			HBsAg+VE	NO	NORMAL	NIL	Dhavaman	VIRAL	LN/EPI				A/MCH	2.6		_		NO	NO
NEG			HBsAg+VE	NO	NORMAL	NIL	Vembu	VIRAL	LN/EPI		_	T	A/MCH	2.7	_	_	_	NO	NO
NEG			HCV+VE	NO	NORMAL	NIL	Rajeswari	VIRAL	LN/EPI		_	T	A/MCH	2.7	_	_		NO	NO
NEG			HBsAg+VE	NO	NORMAL	NIL	Maheshwa	VIRAL	LN/EPI		-	T	A/FCH	2.5		_	_	NO	NO
NEG			HBsAg+VE	YES	NORMAL	/++	Tamillakky	VIRAL	LN/EPI		3PC+12FFP	T	A/FCH	2.7	_	HEP.ENC		NO	DEATH	VIRAL HEPATITIS
NEG			HBsAg+VE	NO	NORMAL	NIL	Vennila	VIRAL	LN/EPI		1PC+4FFP+6PL	T	A/FCH	2.6	_	_		_	NO	
NEG			NEG	NO	NORMAL	NIL	Manimega	HG			2PC	_	_	_	_	_		YES	NO	
NEG			NEG	NO	NORMAL	++	Sharmilaba	Partial HELLP	LSCS	FD	_	T	A/MCH	2.7	_	_	_	NO	NO	
NEG			NEG	NO	NORMAL	NIL	Kavitha	HELLP	LSCS	DIVC	6FFP	PT	A/MCH	2.1	YES	_		NO	NO	
NEG			HBsAg+VE	NO	NORMAL	/+++	Ilakkiya	VIRAL	LSCS	PLSCS	1PC	T	A/MCH	2.6	_	_		NO	NO	
NEG			NEG	NO	NORMAL	NIL	Suganya			NVD	1PC	T	A/MCH	2.7	_	_	_	NO	NO	
NEG			NEG	NO	IUGR	/+++	Selvi	SPE/HELLP	LSCS	NRNST	_	PT	A/FCH	2.1	YES	_		NO	NO	
NEG	↑↑	↑↑	NEG	YES	IUD	NIL	Deivanai	AFLP	LN/EPI		12PC+20FFP+12PLA	PT	D/MCH	1.7		ATONIC		NO	DEATH	HELLP/DIVC
NEG			HBsAg+VE	NO	NORMAL	NIL	Namitha	VIRAL	LSCS	PLSCS	4FFP	T	A/MCH	4.5	YES	ATONIC		NO	NO	
NEG			NEG	NO	NORMAL	NIL	Barshanba	SPE/HELLP	LSCS	OLIGO	4FFP	T	A/MCH	3.5	_	_	_	NO	NO	
NEG			NEG	NO	NORMAL	NIL	Kowsalya		LSCS	FD	_	_	A/MCH	3.3	_	_	_	NO	NO	
NEG			NEG	NO	NORMAL	+++	Soundarya	HELLP	LN/EPI		_	T	A/FCH	2.6	_	_	_	NO	NO	
NEG			HBsAg+VE	NO	NORMAL	NIL	Devika	VIRAL	LN/EPI		_	T	A/FCH	2.6		_	_	NO	NO	
NEG			HBsAg+VE	NO	NORMAL	NIL	Keerthiga	VIRAL	LSCS	MCPD	2PC	T	A/MCH	3.7		ATONIC		NO	NO	
NEG			HBsAg+VE	NO	NORMAL	NIL	Jeya	VIRAL	LN/EPI		_	T	A/MCH	2.8		_	_	NO	NO	
NEG	↑↑	↑↑	HAV+VE	NO	NORMAL	/++	Saranya	VIRAL	LN/EPI		5PC+10P+25FFP	T	A/MCH	2.1	YES	ATONIC	_	IMCU	AMA	
NEG			HBsAg+VE	NO	IA	NIL	Banumathı	VIRAL	MVA	_	2PC	_	_	_	_	_	_	_	_	
NEG			HBsAg+VE	NO	NORMAL	NIL	Vembu	VIRAL	LSCS	CPD	_	T	A/FCH	3.1		_	_	NO	NO	
NEG			NEG	NO	IUGR	+++	Dhanalaksı	SPE/HELLP	LSCS	AKI	1PC	T	A/FCH	2.1	_	_	_	YES	NO	
NEG	↑↑	↑↑	HBsAg+VE	NO	IUD	++	Kala	VIRAL	LSCS	AKI	4FF+2PC+6P+PL	PT	D	1.8	_	ATONIC	_	YES	DEATH	AFLP
NEG			NEG	NO	NORMAL	NIL	Dhanalaksı	_	RPTLSCS	PLSCS	_	T	A/MCH	3.9		_	_	NO	NO	
NEG			NEG	NO	NORMAL	NIL	Jeevitha	_	RPTLSCS	PLSCS	1PC	T	A/FCH	2.8		_	_	NO	NO	
NEG			NEG	YES	NORMAL	NIL	Jenifer	HELLP	MVA	_	3PC	_	_	_	_	AKI	_	_	DEATH	AKI
NEG			HBsAg+VE	NO	NORMAL	NIL	Jeevitha	VIRAL	LN/EPI	_	_	T	A/FCH	2.6	_	_	_	NO	NO	
NEG			NEG	NO	NORMAL	+++	Ilakkiya	HELLP	LSCS	UF CX	4PC+4PL+2FFP	T	A/FCH	2.6	_	_	_	NO	NO	
NEG			HBsAg+VE	NO	NORMAL	NIL	Anjammal	VIRAL	ELLSCS	PLSCS	_	T	A/FCH	2.5	_	_	_	NO	NO	
NEG			NEG	NO	NORMAL	++	veeramma	SPE/HELLP	LN/EPI	_	4PC+4FFP+6PL	T	A/MCH	2.8	_	ATONIC	_	NO	NO	
NEG			HBsAg+VE	NO	NORMAL	NIL	Radhika	VIRAL	ELLSCS	CPD	_	T	A/FCH	2.5		_	_	NO	DEATH	AFLP
NEG			NEG	NO	IUGR	++	Vetriselvi	SPE/HELLP	LN/EPI	_	2PC	T	DEAD/M	2.6	_	_	_	NO	NO	
NEG			NEG	NO	NORMAL	NIL	Deepa	HELLP	LSCS	FD	4FFP+4PL	T	A/MCH	3.1	_	_	_	NO	NO	
NEG			NEG	NS	SPLM	NIL	Gowthami	HELLP	LSCS	CPD	3PC+4FFP+4PL	T	A/FCH	3.1	_	_		NO	DEATH	HELLP
+	↑↑	↑↑	NEG	NO	IUGR	+++	Suganya	INTRAHEPATIC CHOLESTASIS	LSCS	FD	4PC+14FFP+11PLAT	T	A/FCH	2.9	_	ATONIC	_	NO	NO	
NEG	↑↑↑	↑↑	NEG	NO	NORMAL	NIL	Nathiya	HELLP/APECLAMPSIA	M.HYSTEROTOM	AE/FI	1PC	PT	A/FCH	750GMS	_	_	_	_	_	
NEG			NEG	NO	NORMAL	NIL	Shanthy	HYPEREMESIS	MVA/ICA	_	2PC	_	_	_	_	_	_	_	_	
NEG			HBsAg+VE	NO	NORMAL	NIL	Vembarasi	VIRAL	LN/EPI	_	_	T	A/MCH	2.5		_	_	NO	NO	
NEG			HBsAg+VE	NO	NORMAL	NIL	Manjula	VIRAL	LN/EPI	_	_	T	A/FCH	2.7	_	_	_	NO	NO	
NEG			NEG	NO	IUD	NIL	Fathima be	HELLP/AKI	ABD	_	1PC+4FFP	PT	D/FCH	1.4	_	ABRUPTION	_	IMCU	NO	
NEG			HBsAg+VE	NO	NORMAL	NIL	Sarasu	VIRAL	LN/EPI	_	_	T	A/MCH	2.3	_	_	_	NO	NO	
NEG			NEG	NO	NORMAL	NIL	Devi	HELLP	SE	_	4PC	PT	D/MCH	700	_	_	_	NO	NO	
NEG			NEG	NO	NORMAL	NIL	Soundaran	HELLP	LSCS	Partial HELLP	1PC+4FFP	PT	A/FCH	3.4	_	_	_	NO	NO	
NEG			NEG	NO	NORMAL	IUGR	Pavithra	HELLP	LSCS	HELLP	1PC+4PL	PT	A/MCH	1.7	_	_	_	NO	NO	
NEG			NEG	NO	NORMAL	+++	Boopathy	HELLP/VIRAL	LSCS	PLSCS	1PC+8FFP	T	A/FCH	2.4	_	DIVC	_	_	_	
NEG			HBsAg+Ve	NO	NORMAL	IUGR	Selvarani	HELLP/VIRAL	LSCS	MSAF	_	PT	A/FCH	2.6	_	_	_	NO	NO	
NEG			HBsAg+VE	NO	NORMAL	NIL	Tamilarasi	VIRAL	LN/EPI	_	_	T	A/FCH	2.6	_	_	_	NO	NO	
NEG			HBsAg+Ve	NO	NORMAL	NIL	Nagalaksh	HELLP	LSCS	FD	3PC+6FFP	T	A/FCH	2.2		_	_	_	NO	
NEG			NEG	NO	NORMAL	NIL	Lakshmi	HELLP	LSCS	FI	_	T	A/MCH	2.6	_	_	_	NO	NO	
NEG			NEG	NO	NORMAL	NIL	Banu	HELLP	LSCS	HELLP	1PC	T	A/FCH	2.8	_	_	_	NO	NO	
NEG			NEG	NO	NORMAL	NIL	Nandhini	VIRAL	LN/EPI	_	_	T	A/FCH	2.7	_	_	_			
NEG			HBsAg+ve	ND	NORMAL	NIL														